SEXUAL DYSFUNCTION
IN
MALE DIABETICS

THESIS
FOR
DOCTOR OF MEDICINE
(MEDICINE)

BUNDELKHAND UNIVERSITY,
JHANSI

1984   **ANIL KUMAR SACHDEVA**
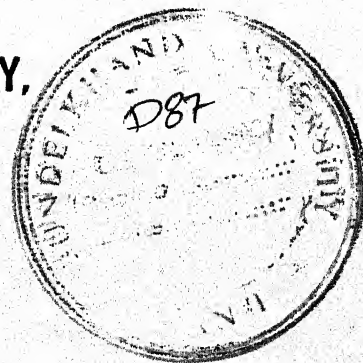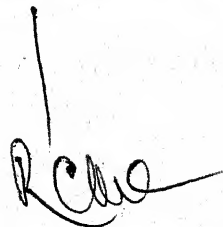
C E R T I F I C A T E

Certified that the research work entitled "SEXUAL DYSFUNCTION IN MALE DIABETICS" which is being submitted as thesis for M.D. (MEDICINE) examination of Bundelkhand University, 1984, by Dr. Anil Kumar Sachdeva has been carried out in the department of Medicine.

He has put in the necessary stay in this department as per University regulations.

(R.C. ARORA)
M.D.,
Professor and Head,
Department of Medicine,
M.L.B.Medical College, Jhansi.

C E R T I F I C A T E

This is to certify that the research work entitled "SEXUAL DYSFUNCTION IN MALE DIABETICS" which is being submitted as thesis for M.D. (MEDICINE) examination of Bundelkhand University 1984, has been carried out by Dr. Anil Kumar Sachdeva under my supervision and guidance . The techniques described were undertaken by the candidate himself.
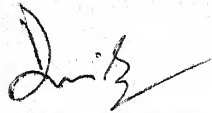
(R.C. ARORA)

M.D.,

Professor and Head

Department of Medicine,

M.L.B. Medical College and

Hospital, Jhansi

C E R T I F I C A T E

Certified that the present research work entitled "SEXUAL DYSFUNCTION IN MALE DIABETICS" was conducted by Dr. Anil Kumar Sachdeva under our direct supervision and guidance. The techniques and statistics mentioned in the thesis were actually undertaken by the candidate himself. His observations have been checked by us regularly.
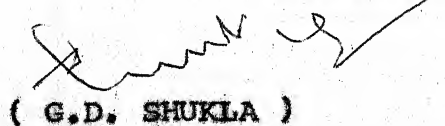
(D.N. MISHRA)

M.D.,

Reader in Medicine,

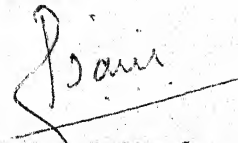M.L.B.Medical College, Jhansi

SUPERVISOR

(G.D. SHUKLA)

M.D.,

Lecturer in Psychiatry,

M.L.B.Medical College,

Jhansi

CO-SUPERVISOR

(P.K. JAIN)

M.D.,M.N.A.M.S.,

Lecturer in Medicine,

M.L.B.Medical College,

Jhansi

CO-SUPERVISOR

ACKNOWLEDGEMENTS

ACKNOWLEDGEMENTS

It is a matter of great privilege to acknowledge my deepest sense of gratitude to my venerable teacher Dr. D.N. Mishra, M.D., Reader in Medicine, under whose condign guidance I had an opportunity to work. I feel highly indebted to him for having freely banked upon his profound knowledge and resources. The present work bears at every stage the impression of his wise and concrete suggestions, careful and reasoned criticisms and meticulous attention.

My respectful and sincere thanks are due to Prof. R.C. Arora, M.D., Head of the Department of Medicine, for giving me the opportunity to work. I am proud of having worked under him. He has been a constant source of encouragement, guidance and paternal bestowments to me.

I find words inadequate to express my feelings of sincere thanks and deep respect to Dr. G.D. Shukla, M.D., Head of the Department of Psychiatry, for his utmost guidance and untiring help during the entire course of the study. His

enthusiasm and energy were enviable. He was also very kind to go through every detail of the manuscript with constructive and critical suggestions. It has been due to his efforts that the thesis has achieved its final shape and is being submitted in time.

I am equally grateful to Dr. P.K. Jain, M.D., M.N.A.M.S., Lecturer in Medicine, who has been kind enough to beckon me to this work. He benefitted me by his timely and valuable suggestions during the course of study.

I also wish to put on record my heart-felt gratitude to Dr. Navnit Agarwal, M.D., Lecturer in Medicine; Dr. Praveen Kumar, M.D., Dip. Card., D.M., Lecturer in Cardiology and Dr. B.L. Verma, Ph. D., Statistician-cum-Lecturer in S.P.M., who were always ready to extend their co-operation during the period of study.
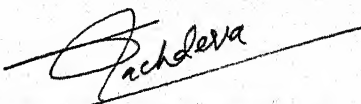
I appreciate the co-operation of the Librarians of National Medical Library, New Delhi and S.N. Medical College, Agra who helped me in collection of literature for the present study.

I dedicate this work to the memory of my late father, Shri V.R. Sachdeva, which was the source of inspiration and solace to me in the execution of this cumbersome task.

I have no words to express my gratitude to my mother and sisters for their constant moral support and for extending a helping hand beyond all limits and expectations.

I wish to accord my thanks to Mr. K. Lal for typing the manuscript with cheerful efficiency.

Finally, I am deeply obliged to the subjects of this study whose co-operation was but essential for the success of this work.

(ANIL KUMAR SACHDEVA)

Dated : 30th May ,1983

C O N T E N T S

INTRODUCTION

Sexual disturbances and diabetes mellitus are both exceedingly common maladies in day to day clinical practice. It is therefore only to be expected that the two could occur together in a sizeable number of patients even if there was no cause and effect relation between them. However, several studies have demonstrated that sexual disorders seen in the diabetics differ both quantitatively and qualitatively from those occurring in the general population (Ellenberg, 1971; Kolodny et al, 1974, 1979 and Krosnick and Podolsky, 1981). Most of the workers have found impairment of sexual functions in around fifty percent of diabetics, the frequency being more than five times higher than that met with in the general population (Rundles, 1945; Rubin and Babbott, 1958; Schoffling et al, 1963; Ellenberg, 1971; Kolodny et al, 1974; Herman et al, 1978 and Krosnick and Podolsky, 1981). This difference between diabetics and non-diabetics has been recorded in all the age groups (Rubin and Babbott, 1958; Frank et al, 1978 and Gebhard and Johnson, 1979).

However, despite the fact that Von Noorden (1903) and Naunyn (1906) recognized and commented upon the frequently impaired sexual functions in diabetics nearly eighty years ago, there have been few planned and systematic studies of the problem, the literature being largely impressionistic. Most of the available reports are beset with methodological tangles, rendering valid conclusions well neigh impossible. Authors have often used terms like libido, potency and fertility imprecisely, vaguely and interchangeably. They have adopted varying case-selection and diagnostic criteria, e.g., studying a small number of cases thoroughly (Lester et al, 1980 and Fairburn et al, 1982) or large number of cases rather superficially (Ellenberg, 1971; Campbell and McCulloch, 1979 and Alam et al, 1981) or only diabetics with impotence (Fairburn et al, 1982) or only diabetics with vasculopathy or some other complication (Herman et al, 1978). Or else they have used "impotence" as an all embracing term to cover all the sexual disturbances, erectile as well as ejaculatory. The results, quite understandably, vary widely from one study to the other, ranging from 10 percent (Gahlaut and Sharma, 1982) to 84 percent (Khandelwal et al, 1981). Further most of the studies have been uncontrolled making it difficult

to evaluate their results in statistical term
(Lester et al, 1980).

Till recently, it was believed that
impotence in diabetics followed one of the two
classical patterns (Kolodny et al, 1979). In the
first, erectile failure was felt to occur in the
context of poor diabetic control. There was complete
loss of interest in sex due to general malaise and
the symptoms reversed once the patient's general
physical condition improved with the control of
diabetes (Oakley, 1949; Keen, 1959 and Kolodny et
al, 1979). The second form was said to be characteristic
of diabetes and was held to be progressive and
irreversible. All erections were affected including
those obtained spontaneously and on waking up and
there was no associated loss of sexual interest
(Podolsky, 1971; Jadzinsky et al, 1973 and Faermen
et al, 1980).

Further, diabetic impotence has been held
to be mainly organic in origin, due most probably
to autonomic neuropathy (Ellenberg, 1971; Kolodny
et al, 1974, 1979 and Krosnick and Podolsky, 1981),
though psychological (Hosking, 1979), vascular
(Herman et al, 1978), endocrinological (Wagner et al,
1942; Schoffling et al, 1963 and Moses et al, 1980)

and other factors like drugs, alcohol and infections (Masters and Johnson, 1970; Alam et al, 1981 and Krosnick and Podolsky, 1981) have received emphasis from some quarters.

Recently, these simplistic "either or" views have come under strong attacks (Fairburn, 1981; Fairburn et al, 1982 and Tattersall, 1982). These authors have seriously questioned the validity of the two classical patterns of diabetic sexual disturbances referred to above. It has been suggested that patients with "intermediate features" far outnumber those with classical ones (Schiavi and Hogan, 1979 and Scott et al, 1980). Similarly, aetiological hypotheses dividing the cases into "organic" and "functional" and the former into "neurogenic", "vascular" and "endocrinal" are no longer firmly held. It is the common consensus that sexual functioning in the human male represents a complex and dynamic interaction between psychic and somatic determinants. Sexuality is influenced by several conscious and unconscious factors, by such physiological factors as neurological, vascular and endocrinological ones and by sociocultural factors like education and prevalent sexual mores and attitudes. The sexual process is very sensitive and may be disturbed by any one of them.

In view of the theoretical as well as clinical importance of the problem and the prevailing controversies regarding its various aspects in the current literature, it has been thought worthwhile to undertake the present work with the following objectives :-

1. To find out the frequency and pattern of sexual disturbances in a group of male diabetics as against those in a group of age-matched controls.

2. To relate such disturbances with various aspects of diabetes.

3. To highlight and discuss the results in the light of available relevant literature.

REVIEW OF LITERATURE

R E V I E W   O F   L I T E R A T U R E

HISTORICAL AND GENERAL ASPECTS :

Diabetes mellitus has been known to mankind from the times immemorial. It has been described by the physicians of the ancient civilizations like Greece, Egypt, India and China. The Egyptian Papyrus of Ebers (1500 BC), a medical compilation, has made the mention of this malady with polyuria. The Greeks, who knew about its prominent manifestations of persistent polyuria, named the disease "diabetes" (passing like a fountain or through a siphon). Aretaeus of Cappadocia (30-90 A.D.) described the disease as a "melting down of the flesh and limbs into the urine". In India, the disease was better understood. Charaka (Second century A.D), in his "Charak Samhita", has mentioned the sweetness of urine in addition to the symptom of polyuria. Charaka collected material for his compilation from much earlier works of Agnivesa, who based his writings on the teachings of Atreya, who lived in the sixth century B.C. The Indian surgeon, or rather the father of Indian surgery, Sushruta, in 500 A.D.,

described the disease as "Madhumeha" (passing honey urine) with symptoms of thirst, foul breath, voracious appetite and langour (Nayyar, 1966).

Thomas Willis in 1675 proved that sugar passed in the urine of the diabetic patient. In 1889, Minkowski produced diabetes experimentally by removing the pancreas from a dog. Langerhans described his islets in 1869, and the hypothetical internal secretion of pancreas was named "insulin" long before it was isolated. Its final extraction in 1921 by Banting, then a young orthopaedic surgeon, and Best a junior graduate assistant was one of the great medical events of the century (Bomford and Mason, 1973).

The disease is usually defined as a chronic disorder of carbohydrate metabolism characterised by hyperglycaemia and glycosuria due to relative or absolute deficiency of insulin (Joslin, 1959). The disease manifests as a series of hormone-induced metabolic abnormalities, by long term complications, and by micro-vascular lesions, demonstrable by electron microscopy (Foster, 1980).

Diabetes is perhaps the commonest serious metabolic disease of human beings. It is uncertain

whether its incidence has fluctuated through the ages since there has been no clear cut definition of the disease. Surveys have shown differing figures for its prevalence, depending upon the techniques and criteria adopted (Pyke, 1968). Two large population based studies for the prevalence of diabetes include those conducted by Wilkerson and Krall (1947) at Oxford, Mass, USA with a prevalence rate of 1.40 percent and the diabetes survey in Bedford, England by Butterfield (1964) with a prevalence rate of 1.03 percent. Till recently, it was a common belief that diabetes was less common in India and that if it occurred, its severety was less (Mills, 1930 and De Zoysa, 1951). This view had been held mainly because of the paucity of epidemiological data. Population based studies undertaken in the past two decades have conclusively demonstrated that the disease occurs with equal or even higher frequency in this country, the reported prevalence rates ranging from 1.9 percent to 4.0 percent (Berry et al, 1966; Patel et al, 1966; Satyanarayan et al, 1966; Gupta et al, 1970; Tripathy, 1971 and Ahuja et al, 1972).

Diabetes mellitus is a chronic disease of obscure aetiology. Its treatment, therefore, is essentially symptomatic, i.e., it merely lowers the

blood sugar level without reversing the basic
pathophysiological defect(Joslin, 1959). The
disease, quite understandably has several long-term
and far reaching complications, involving almost
every organ and system of the body. Some of the
important ones include : vasculopathy (Clawson
and Bell, 1949; Bell, 1952; Goldenberg et al, 1958;
Ostrander et al, 1965; Malins, 1966; and Foster,
1980), neuropathy (Rundles, 1945; Collens et al,
1950; Laurence and Locke, 1963; Mayne, 1965;
McFadgean and Yeung, 1968; Rackow, 1968; Bhu and
Bharadwaj, 1969; Nies, 1972; Wingrade, 1972; Viswanath
et al, 1976; Mohan et al, 1980 and Noronha et al, 1981),
retinopathy and other ocular manifestations (Paterson
et al, 1957; Malins, 1968 and Foster, 1980),nephropathy
(deWardner, 1969) and sexual disturbances (Ellenberg,
1971; Kolodny et al, 1974; Krosnick and Podolsky,
1981; Khandelwal et al, 1981; Gahlaut and Sharma,
1982 and Tattersall, 1982). Since the present work
deals with sexual disturbances in male diabetics,
the review henceforth will be limited to their
various aspects.

Although as early as the first decade of
the present century, Von Noorden (1903) and Naunyn
(1906) wrote that impotence was one of the commonest

symptoms of diabetes, the subject has largely been ignored as is evident from the paucity of data having direct bearing on it, particularly from our country (Alam et al, 1981 and Gahlaut and Sharma, 1982). Diabetic clinics are often so busy and open that the patients feel rather inhibited in talking about such intimate problems as those pertaining to sex (Tattersall, 1982). Or else, the patients feel their sexual problems to be unrelated to diabetes and hence do not volunteer these informations unless asked for (Ellenberg, 1971). It was most probably because of this reason that Joslin (1959) commented that sexual disturbances were exceedingly rare in diabetics.

As against this, there are several reports documenting frequent occurrence of sexual disturbances in patients of diabetes mellitus (Rundles, 1945; Simpson, 1950; Rubin and Babbott, 1958; Keen, 1959; Klebanow and MacLeod, 1960; Montenero and Donatone, 1962; Schoffling et al, 1963; Ellenberg, 1971; Kolodny et al, 1974; Hastings, 1975; Marmor, 1975; Campbell and McCulloch, 1979; Hosking, 1979; Moses, 1980; Tattersall, 1982). The reported frequency of sexual disturbances varies widely. Thus while Gahlaut and Sharma (1982) reported an overall rate of 10 percent

(24 percent in 21-40 years age group) and Viswanath
~~Alam~~ et al (1976), Noronha et al (1981), ~~Viswanath~~
Alam et al (1981) and Campbell and McCulloch (1979)
reported rates around 35 percent; Khandelwal et al
(1981) found as high as 84 percent of their diabetic
patients to be having one sexual disturbance or the
other. Majority of workers have reported rates around
50 percent (Rundles, 1945 ; Rubin and Babbott, 1958 ;
Schoffling et al, 1963 ; Kolodny et al, 1974 ; Herman
et al, 1978 and Krosnick and Podolsky, 1981). Rubin
and Babbott (1958) and Marmor (1975) felt that the
rates of sexual disturbances in diabetics were about
5 times of those for the general population.

In the light of these figures, it is rather
surprising that, in a recent study, Lester et al
(1980) reported the overall incidence of sexual
disturbances in diabetics to be just 23 percent-a
figure that was alomost same (20%) as that for an
age and sex matched control group of non-diabetics.
It is difficult to account for such marked differences
in the figures. One factor could be the varying
criteria adopted for the diagnostic evaluation of
diabetes mellitus. Another factor might be the
varying intensity of the interrogation to unearth

the symptoms of sexual disturbances and varying
significance assigned to them. Since the patients
seldom come out, on their own, with sexual
disturbances and since many of them might feel it
only natural to lose sexual prowess with age and
may take it to be rather silly and absurd to talk
about to a doctor unless the physician undertakes
an attitude of active searching and sympathetic
listening he is likely to miss many genuine cases
of sexual disturbances or to undermine their
significance in the overall evaluation of the case
(Tattersall, 1982).

PATTERN OF SEXUAL DISTURBANCES IN DIABETES MELLITUS :

Most of the workers have used the term
"impotence" to denote the sexual disturbances
encountered in diabetic males (Rundles, 1945 ;
Rubin and Babbott, 1958 ; Klebanow and MacLeod,
1960 ; Schoffling et al, 1963 ; Ellenberg, 1971 ;
Cooper, 1972 ; Herman et al, 1978 ; Campbell and
McCulloch, 1979 ; Krosnick and Podolsky, 1981 and
Gahlaut and Sharma, 1982). This is surprising in
view of the fact that impotence is just one of the
several possible sexual dysfunctions. These authors
have probably used the word "impotence" as an
all-embracing term to include all the types and
degree of sexual dysfunctions. This certainly is
an over simplification of the real state of affairs

since, as reported by Kolodny et al (1974), Khandelwal et al (1981), Alam et al (1981) and Fairburn et al (1982), diabetic males are subject to a wide variety of sexual disturbances. Three important and rather classical disturbances are the following :-

1- Impotence : "Failure to attain and/or sustain penile erection for a satisfactory intercourse on most of the attempts" (Hastings, 1975).

2- Premature ejaculation : "Inability of the male to control ejaculation long enough to satify his partner in atleast 50 percent of their coital opportunities, when their is no female dysfunction" (Masters and Johnson, 1970) or "Inability of the male to exert voluntary control over the ejaculatory reflex" (Kaplan et al, 1974) or "A condition wherein the male arrives at orgasm and ejaculates before he wishes to do so" (Hastings, 1975).

3- Retrograde ejaculation : "A condition wherein a male has orgasm but the ejaculate, instead of being discharged per urethra, is forced backwards into the urinary bladder (Ellenberg and Weber, 1966 ; Kolodny et al, 1974 and Krosnick and Podolsky, 1981).

Besides these there are some relatively less well known though equally distressing abnormalities (Fairburn et al, 1982 and Tattersall, 1982).

1:    Trickling, dribbling or seepage of the semen without orgasm. Here the semen starts dribbling out just after or even without erection.

2:    Frequent erections without any sexual thoughts or excitement. With passage of time impotence supervenes in these cases.

3:    Penile deformity in form of upward bending midway along the shaft so that the penis resembles a smoker's pipe in profile.

IMPOTENCE :

Impotence, i.e., failure to have erections strong enough for a satisfactory sexual intercourse

is undoubtedly the commonest and the most distressing
sexual disturbance occuring in the diabetic males.
Depending upon the case material and upon the
definitions of diabetes and impotence adopted,
the latter has been found to occur in around 50
percent of the cases (Rundles, 1945 ; Rubin and
Babbott, 1958 ; Schoffling et al, 1963 ; Ellenberg,
1971 ; Kolodny et al, 1974 ; Herman et al, 1978 and
Krosnick and Podolsky, 1981). However, some workers
have reported rather low figures (10 percent by
Gahlaut and Sharma, 1982 ; 23 percent by Lester et
al, 1980 ; 25 percent by Noronha et al, 1981 ; 33
percent by Alam et al, 1981 and 35 percent by
Campbell and McCulloch, 1979), while Khandelwal
et al (1981) found 84 percent of their cases to
be impotent. These figures are several times of
those reported for the general population by
Kinsey et al (1948) and Masters and Johnson
(1970). The higher figures in diabetics compared
to those for the general population are seen in
all the age groups (Rubin and Babbott, 1958 ;
Frank et al, 1978 and Gebhard and Johnson, 1979).
Lester et al (1980), on the other hand found no
difference between diabetics and non-diabetics as

regards the frequency of impotence, the rates being
23 percent and 20 percent respectively, in the two
groups.

Any review of sexual functions in diabetic
males must contend with the varying case-selection
criteria and the intensity of case work up. Workers
have studied only impotent diabetics (Fairburn et al,
1982), or only diabetics with vasculopathies (Herman
et al, 1978). Further, they have either studied a
large case material rather superficially (Ellenberg,
1971 ; Campbell and McCulloch, 1979 and Alam et al,
1981) or a very small group of patients very thoroughly
(Lester et al, 1980 and Fairburn et al, 1982). There
is no population based epidemiological data, all the
work having been carried out on patients attending
diabetic facilities. The cases therefore are likely
to be established and severe ones and the information
gathered mostly retrospective and anecdotal. Lastly,
most of the studies have been uncontrolled thus
making it difficult to evaluate the resluts in valid
statistical terms (Lester et al, 1980).

Two types of diabetes related impotence
have classically been described (Kolodny et al,
1979). In the first, failure of erection occurs

in the context of poor diabetic control. Erectile
failure and loss of interest in sex accompany the
general malaise and the symptoms reverse once the
patients' general physical condition imporoves with
the control of diabetes. This type of impotence is
non-specific and is found in other debilitating
conditions as well (Oakley, 1949 ; Keen, 1959 and
Kolodny et al, 1979). Impotence here is believed
to be mainly due to histochemical factors such as
sorbitol and water accumulation in the nerve fibers
caused by poor blood glucose control (Campbell and
McCulloch, 1979 and Faerman et al, 1980). The
second form of impotence is said to be characteristic
of diabetes. It is thought to be the result of a
physical process and is generally described as
progressive and irreversible. All erections are
affected, including those obtained spontaneously
and on waking up, and is associated with no loss
of sexual interest. Here the disturbance is perhaps
due to histologic changes in the autonomic fibers
of corpora cavernosa (Podolsky, 1971 ; Jadzinsky
et al, 1973 and Faerman et al, 1980). The subject
of aetiology of impotence in diabetes will be
discussed in greater detail in subsequent pages.

The validity of these clinical stereotypes
has recently been questioned (Fairburn, 1981 ;
Fairburn et al, 1982) and it has been suggested
that many patients show "intermediate features"
(Schiavi and Hogan, 1979 ; Scott et al, 1980 and
Tattersall, 1982). Furthermore, there have been
reports of successful use of sex therapy in the
treatment of diabetic impotence, a supposedly
irreversible condition hithertofore (Renshaw,
1975, 1979).

A rather striking and oft-reported feature
of diabetic impotence is the persistence of libido
(sexual desire) despite absence of potency (Rubin
and Babbott, 1958 ; Ellenberg, 1971 ; Kolodny et
al, 1974 ; Lester et al, 1980 ; Moses et al, 1980
and Krosnick and Podolsky, 1981). This was particularly
so in the younger patients (Rubin and Babbott, 1958).
However, there are few reports that libido does show
a decline (Alam et al, 1981 ; Khandelwal et al,1981
and Fairburn et al, 1982). Alam et al (1981) reported
altered libido in 70 percent of their cases and
Fairburn et al (1982) in 44 percent. This could be
because of a substantial number of uncontrolled cases
in these studies. Unfortunately, because there is no

objective measurement of sexual interest, data are mostly anecdotal. Aside from rare endocrine problems, the major cause of decreased libido is depression and there is no evidence to suggest that diabetics are depressed more often as a group than the non-diabetics (Campbell and McCulloch, 1979; Krosnick and Podolsky, 1981 and Gahlaut and Sharma, 1982). A more likely cause of the apparent loss of libido could be the erectile impotence itself. The male diabetic who suffers from erectile failure may attempt to conceal it by feigning a "lack of interest" in sex (Ellenberg, 1971; Krosnick and Podolsky, 1981). On close questioning Ellenberg (1971) found libido to be virtually unchanged in many of his cases despite impotence of more than 10 years duration.

Attempts have been made to relate the frequency and intensity of the impotence with various aspects of diabetes mellitus and it would be worthwhile to review them briefly.

Age :

Even in normal population, sexual vigour declines with age (Kinsey et al, 1948). In their sample the incidence of impotence was practically nil at the age of 35 years as against 77 percent at 80.

In diabetics the incidence becomes manifold, which is true for all the age groups (Rubin and Babbott, 1958; Klebanow and MacLeod, 1960; Schoffling et al, 1963; Kolodny et al, 1974; Marmor, 1975; Alam et al, 1981 and Gahlaut and Sharma, 1982). Rubin and Babbott (1958) found the incidence to be 25 percent in the 30-34 years age group, 54 percent in 50-54 years age group and 75 percent in the 60-64 years age group. These figures were 2-5 folds greater than the corresponding figures of Kinsey et al (1948). Similarly, Schoffling et al (1963) reported 29 percent under the age of 30 years as against 73 percent beyond the age of 60 years. Alam et al (1981) reported maximum incidence in the fifth decade while Schoffling et al (1963) found impotence to be commonest in the seventh decade of life.

Duration of diabetes :

Authors differ in their observations regarding the effects of the duration of diabetes on the incidence of impotence. Thus, while Kolodny et al (1974), Ellenberg (1971) and Moses et al (1980) found no effect of duration of diabetes on incidence or duration of impotence, several others have reported a direct relation between the two (Schoffling et al,

1963; Marmor, 1975; Khandelwal et al, 1981 and
Gahlaut and Sharma, 1982). Rubin and Babbott (1958)
found the incidence of impotence to be 70 percent in
diabetics with upto one year duration of the disease,
43 percent in patients with a duration of 1-5 years
and 45 percent in those with a disease having lasted
more than 5 years. The authors concluded that the
incidence of impotence did not increase with a greater
duration of the disease. They felt that the rather
high (70 percent) incidence in men with a recent onset
of diabetes might have been due to the fact that many
of them had just been found to be diabetics and their
disease often had not yet been well controlled. They
further found that 30 percent of all the those who
became impotent did so within one year of the clinical
recognition of the diabetes and sixty percent within
five years. These observations were later supported
by others who found that impotence occurring early
in the course of diabetes was often due to general
malaise and improved with the control of diabetes
(Kolodny et al, 1974; Campbell and McCulloch, 1974
and Faerman et al, 1980). Moreover, in 7-15 percent
of the cases, sexual impotence has been found to ante-
date the clinical recognition of diabetes and to be
the first symptom that sent the patient to a physician

and thus ultimately led to the discovery of the disease (Rubin and Babbott, 1958; Ellenberg, 1971; Kolodny et al, 1974 and Moses et al, 1980).

Schoffling et al (1963) have reported contrary findings. Amongst 314 male diabetics whose diabetes developed after 60, 160 (51 percent) had impotence. Mean duration of diabetes was 9.3 years in the impotent group as against just 4.3 years in the potent group. Khandelwal et al (1981) found the incidence and severity of impotence to be proportional to the duration of diabetes mellitus. Similarly, Gahlaut and Sharma (1982) found a statistically significant relation between the duration of diabetes and the incidence of impotence, the latter being 6.6 percent in diabetics with upto 2 years duration as against 18 percent in those with a duration of 6 years the overall incidence of impotence in their series being 10 percent.

Severity of diabetes :

Gahlaut and Sharma (1982) reported a statistically significant relation between the incidence of impotence and the severity of diabetes expressed in terms of fasting blood sugar levels.

Three percent of those with levels upto 140 mg percent were impotent while patients with levels beyond 220 mg percent had an incidence of 15.34 percent. This relationship has, however, not been confirmed by other workers who failed to find any clear cut influence of the severity of diabetes interms of carbohydrate tolerance or insulin requirement, on the incidence or severity of impotence (Rubin and Babbott, 1958; Ellenberg, 1971 and Khandelwal et al, 1981). However, Rubin and Babbott (1958) found that poor control of diabetes, with episodes of acidosis or hypoglycaemia was often associated with transient periods of impotence.

Mode and degree of control of diabetes :

Occurrence of impotence has been found to have no relation with the mode of treatment of diabetes-insulin or oral hypoglycaemic agents (Kolodny et al, 1974 and Marmor, 1975). As regards the relation of impotence with the control of diabetes, two patterns have emerged. In the first, where impotence results early in the course of diabetes, in the setting of poorly controlled disease, it is a reflexion of the associated malnutrition, general malaise and metabolic

imbalance. In such cases, proper management of metabolic aspects of the disease would restore potency by restoring normal health and vigour (Oakley, 1949; Rubin and Babbott, 1958; Ellenberg, 1971; Kolodny et al, 1974; Hastings, 1975 and Moses et al, 1980). Seventy percent of men with a recent onset of diabetes (less than one year prior to the investigation) were impotent in the series of Rubin and Babbott (1958). Many of them regained potency with proper control of diabetes. Similarly, in the study of Kolodny et al (1974), there were 14 (7 percent) cases where impotence had been the initial manifestation of diabetes, preceding the establishment of diagnosis. Half of these men regained potency following the initiation of antidiabetic therapy.

On the other hand, impotence occurring in a well controlled diabetic is almost always permanent, there being no effective prophylactic or therapeutic measure, except in a few cases of psychogenic impotence which are benefited by psychotherapy (Keen, 1959 and Ellenberg, 1971).

Fertility :

Schöffling et al (1963) reported that wives of impotent diabetics had fewer conceptions and more

miscarriages even before they became impotent. In
many cases wives became pregnant following hormone
therapy after prolonged periods of sterility.
Similarly, Neudorfer (1950) found that wives of
24 out of 85 diabetics were sterile—a rate much
higher than that found in the general population.
Several authors have reported low seminal volume,
sperm count, impaired sperm motility and low seminal
fructose levels in diabetics (Warren and LeCompte,
1952; Klebanow and MacLeod, 1960; Schoffling et al,
1963; Bartak et al, 1975; Moses et al, 1980 and
Khandelwal et al, 1981). Thickening of the basement
membrane of the seminiferous tubules (Schoffling et
al, 1963),cessation of spermatogenesis and hyalinization
of tubules (Warren and LeCompte, 1952), atrophy of
tubules and interstitial fibrosis (Koch, 1910),
calcification of vas deferens (Wilson and Marks,
1951) and sparse distribution of Leydig's cells
(Moses et al, 1980) etc. have been observed on
testicular biopsy of impotent diabetics. Klebanow
and MacLeod (1960) found spermatogenesis to be normal
though sperm motility was low in half of the cases.
However, an overwhelming majority of workers have
found sperm count and motility and testicular biopsy
to be perfectly normal in potent as well as impotent

diabetics (Oakley, 1949; Keen, 1959; Rivarola et al, 1970; Ellenberg, 1970 and Faerman et al, 1972, 1974). Reviewing the subject, Warren and LeCompte (1952) concluded that there was no change in adequately treated and controlled diabetics. Further, Ellenberg (1971) and Schoffling (1970) have conclusively demonstrated that there was no relation between potency and sperm count.

Aetiology of impotence in diabetes mellitus :

The exact pathophysiologic mechanism responsible for impotence in diabetic males is not clear (Rubin and Babbott, 1958; Kolodny et al, 1974; Hosking et al, 1979; Moses et al, 1980 and Alam et al, 1981). Sexual functioning in the human male represents a complex and dynamic interaction between psychic and somatic determinants. Sexuality is influenced by several conscious and unconscious factors, by such physiological factors as neurological, vascular and endocrinologic status and age and by such socio-cultural factors as income, education and the prevailing sexual mores and attitudes. The sexual process is very sensitive and may be disturbed by any of these factors (Hastings, 1975). It must therefore be emphasized that all the causes of

impotence in the non-diabetic population can, mutatis mutandis, lead to impotence in the diabetic male. However, the significance assigned to individual aetiological factors would be very much different. Some of the important causes, having specific bearing upon diabetic impotence will be briefly reviewed.

The causes of impotence in diabetes could be classified as follows (Ellenberg, 1971) :

1. Psychogenic
2. Organic
   - Neuropathy
   - Vasculopathy
   - Endocrinopathy.
3. Drug induced
4. Associated conditions
   - Infection
   - Cardio-vascular disease
   - Renal failure

Psychogenic :

In the general population, impotence is "functional", "psychogenic" or "non-organic" in 90 percent of the cases (Simpson, 1950; Cooper, 1972;

Kolodny et al, 1974 ; 1979 and Hosking, 1979).
However, Montague et al (1979) found only 51
percent to be functional ; some organic cause
could be detected in the remaining. Common sense
dictates that diabetics would be equally or perhaps
even more prone to psychological stresses of life,
the diabetic state in itself being a significant
stress (Kolodny et al, 1979). The complexities of
management of the disease often cause psychological
problems which may lead to marital disharmony.
Further, the knowledge that diabetes may interfere
with potency, on the part of the patient, might
lead to a fatalistic attitude- the first sexual
failure being thus regarded as "the end", with
performance anxiety perpetuating the problem.In
view of these considerations it is quite natural
to expect psychogenic impotence to be more common
in diabetics than in the general population (Strauss,
1950 and Tattersall, 1982). In fact, Hosking (1979)
suggested that diabetic impotence was mostly
psychological.

However, the observed findings are quite
contrary to the above expectations. Most of the
workers have found the diabetic impotence to be

largely (in approximately 90 percent of the cases)
organic - the conclusion being based on the pattern
of symptoms (Rubin and Babbott, 1958; Keen, 1959,
Ellenberg, 1971; Campbell and McCulloch, 1979; Alam
et al, 1981 and Gahlaut and Sharma, 1982). Psychological
impotence usually does not prevent morning erections
or erections from masturbation and the impotence is
selective in nature, i.e., being present only in
certain circumstances or with certain sexual partners.
As against this most of the diabetic impotent men are
unable to experience erections under any circumstances
(Strauss, 1949, 1950; Fisher, 1965; Karacan, 1966 ;
Johnson, 1968 and Ellenberg, 1971). Further more, in
many instances, the men became impotent even before
they knew that they had diabetes, impotence being the
presenting symptom whose elaborate work up led to the
diagnosis of diabetes (Rubin and Babbott, 1958 ;
Ellenberg, 1971 ; Kolodny et al, 1974 and Moses et al,
1980). Alam et al (1981) found psychological factors
which could be responsible for impotence in only 16
percent of their cases. Further, Campbell and McCulloch
(1979) found psychiatric symptoms to be rather rare
(only 24 out of 541) while none of the patients of
Gahlaut and Sharma (1982) had any psychiatric features
which could account for the impotence.

Thus it could be concluded that, in general, psychological factors are of very little significance in diabetic impotence. They could be important in the impotence occurring early in the course of diabetes. In the case of impotence occurring in the long standing diabetes, the psychological factors are over-shadowed by organic ones.

Neuropathy :

An underlying neuropathic factor in the diabetic impotence was suggested by the facts that potency depends upon the integrity of the autonomic nervous system and that the latter is frequently involved in diabetic neuropathy. This hypothesis was further encouraged by several studies that indicate an uniform association between impotence and dysfunction of the incipient, asymptomatic neurogenic bladder (Keen, 1959, Ellenberg and Weber, 1967 and Ellenberg, 1980). Malins and Mayne (1965) and Whalen et al (1969) found all cases of diabetic enteropathy to be impotent. Autonomic pathways involved in micturition and erection are identical (Learmouth, 1931). When these nerves are intact, bladder function is normal. Since there is no direct method of objectively measuring impotence,

neurogenic bladder studies (Cystometric recordings, measurement of vesical residual urine, bladder capacity and cystoscopy) are performed-the assumption being that involvement of these nerves would be reflected simultaneously by abnormalities in both areas. Evidences favouring such an association have been overwhelming (Rundles, 1945; Martin, 1953 ; Ellenberg, 1971; Brit. Med. J., 1974 and Kolodny et al, 1974). Not only that, other diseases affecting spinal cord such as tabes dorsalis, syringomyelia, pernicious anaemia, myelitis, multiple sclerosis and spinal cord tumours are almost always associated with impotence (Ellenberg, 1971). Further, the antihypertensives and antidepressant drugs that may lead to impotence are agents that act through or have side effects involving the autonomic nervous system (Kolodny et al, 1979).

Penile erection takes place as a reflex stimulation of the second, third and fourth sacral components of the parasympathetic system (i.e., nervi erigentis), which may be psychic or physical, results in dilation of the penile arteries, increase in blood flow and tumescence of the corpora cavernosa and corpus spongiosum. Erection is thus induced by

active hyperaemia of the arteries which are partially
occluded by longitudinal ridges in the resting state.
With retention of blood, the covernous spaces expand
and thus sustain erection. Detumescence results from
vasoconstriction of the penile arteries with subsequent
decrease of arterial blood flow, diminution of pressure
and release of the compressed veins (Learmouth, 1931;
Bobs and Comarr, 1960 and Weiss, 1972).

Neuropathies, involving peripheral and
autonomic nerves are quite common in diabetics.
Of these, the latter is particularly significant
so far as impotence is concerned. Autonomic neuropathy
has been reported to occur in upto or more than three
fourths of the cases (Collens, 1950; Keen, 1959; Mayne,
1965; Viswanath et al, 1976; Dyrberg et al, 1981 and
Noronha et al, 1981). Further, significantly greater
incidence of neuropathy has been found in diabetic
men with impotence than in non-impotent diabetics
(Ellenberg, 1971; Kolodny et al, 1974; Alam et al,
1981; Khandelwal et al, 1981; Krosnick and Podolsky,
1981 and Ghlaut and Sharma, 1982). Of 118 impotence
diabetics, Ellenberg (1971) found evidence of neuropathy
in 88 percent compared to in only 12 percent of 82
percent diabetics. Kolodny et al (1974) also found

evidence of neuropathy to be significantly higher in a group of impotent (38 percent) than in non-impotent diabetics (21 percent). Faerman et al (1974) demonstrated histological evidence of autonomic neuropathy in the neural fibres of corpora cavernosa.

Sexual impotence have been found to be one of the commonest symptoms of autonomic neuropathy (Bhatia et al, 1976; Viswanath et al, 1976; Mohan et al, 1980 and Noronha et al, 1981). It may, at times be the only symptom (Brit. Med. J., 1974). These evidences suggest that diabetic impotence is, to a great extent, due to neurogenic mechanisms (Ellenberg, 1971 and Faerman et al, 1974). Weiss (1972) in a review of the physiology of penile erection has discussed various anatomical sites of pathology which could account for the problem of impotence. Valve-like structures called "polsters" containing smooth muscle fibers have been described near the corpora cavernosa and are under the control of the autonomic nervous system. Poor neural transmission would disturb the steady state of increased inflow of blood into the erectile tissue and would thus lead to impotence.

However, criticism to the neurogenic theory
of diabetic impotence is not lacking. Kim (1966)
found no evidence of autonomic neuropathy in 8
impotent diabetics (aged 33 - 48 years) on insulin
tolerence - ephedrine exceretion test. Another very
strong evidence against neurogenic theory came from
Fairburn et al (1982) who found no difference in the
frequency of impotence between diabetics with and
without autonomic neuropathy. The ultimate proof of
the neurogenic theory would requir the demonstation
of a specific pathologic, biochemical, enzymatic or
other lesions(s)within the nervous system and the
exclusion of other possible contributory lesions.
Such proof is not available and the evidence in
support of the neurogenic theory, though very strong,
has so far been rather circumstantial (Herman et al,
1978).

Vasculopathy :

Penile erection is haemodynamic phenomenon
controlled by a neurogenic mechanism (Herman et al,
1978). Engorgement of corpora cavernosa and corpus
spongiosum by the blood is the essential mechanism
of normal erection. It is therefore conceivable that

diabetic impotence may be a result of vasculopathy since the latter is an integral part of the pathophysiology of diabetes mellitus (Simpson,1950; May et al, 1969; Gaskell, 1971; Ewing et al, 1973 ; Kolodny et al, 1974; Michal et al, 1974; Abelson, 1975; Herman et al, 1978; Machleder, 1978; Magee and Fried, 1978 ; Hosking, 1978 ; Kolodny et al, 1979 and Krosnick and Podolsky, 1981). Diabetic retinopathy has been found to be a significant correlate of impotence (Tattersall, 1982). Of the diabetics beyond the age of 50 years, only 50 percent of those without retinopathy were impotent as against 100 percent of those with it - a really compelling evidence in favour of vasculopathy as the cause of diabetic impotence.

Endarteritis as a possible aetiological mechanism for diabetic impotence was suggested by Simpson (1950) but appeared to be untenable in young patients without any evidence of vascular disease. Magee and Fried (1978) attributed impotence to the incompetence of deep dorsal vein valves. Occlusive atherosclerotic peripheral vascular disease, affecting the terminal aorta (Leriche syndrome) and the common and internal iliac arteries has been known to lead to impotence (Herman et al, 1978 and Krosnick and

Podolsky, 1981). Gaskell (1971) and Abelson (1975)
reported abnormal penile pulse and blood pressure
in impotent diabetics while May et al (1969), Michal
et al (1974) and Machleder (1978) reported clinical
improvement of impotence after endarterectomy of
the internal iliac artery orifice during aorto -
iliac reconstruction.

Herman et al (1978) carried out translumbar
aortographic studies in 91 patients having leg
ischaemia. Of the 62 non-diabetics, 29 percent were
impotent while of 29 diabetics 59 percent were impotent
the difference being highly significant. The impotent
patients showed a significantly greater stenosis of
internal pudendal arteries than the potent patients -
irrespective of the presence or absence of diabetes.
However, neither diminished femoral pulses not
aortographic evidence of iliac and femoral arterial
stenosis correlated significantly with impotence.
Although all vessels (common iliac, internal iliac
and internal pudendal) showed greater mean stenosis
in the impotent patients, the most consistent
differences were in the internal pudendal vessels
which were the smallest vessels that could be
identified and evaluated arteriographically. It is

conceivable that even smaller vessels are also affected - may be even to a greater extent and that internal pudendal artery stenosis is just a marker of these patients with small vessel disease.

These observations would suggest that vascular lesions may be important in the genesis of impotence. However, their relation to diabetic impotence _per se_ is not strengthened by the available data. The line of evidence suggested by these findings is again circumstantial as is most evidence dealing with the subject of impotence in diabetes. There might be other explanations and an interplay between the vascular lesion and a peripheral neurological lesion cannot be ruled out.

Endocrinopathy :

Endocrinological basis for diabetic impotence has long been espoused. Wagner et al (1942) noted that male juvenile diabetics frequently showed a lag in sexual development manifested by a delayed appearance of pubic and axillary hairs, slow incrementation in testicular size and late voice change. However, the most significant evidence supporting endocrine basis of impotence in diabetics comes from the work of

Schoffling et al (1963). He reported lower than
normal urinary excretion of pituitary gonadotrophins
and normal or slightly raised excretion of 17-ketosteroids
in two thirds of impotent diabetics. One third of his
cases had low sperm counts. Further, they found that
in most of the patients under 40 years of age,
impotence was corrected by a combined therapy with
chorionic gonadotrophins and testosterone. Wives of
many became pregnant after prolonged periods of
sterility. Based on these findings, the authors
suggested that impotence and infertility in male
diabetics was frequently due to hypogonadotrophic
hypogonadism. They could not, however, explain the
mechanism of impaired pituitary gonadotrophic function.
Similarly, Moses et al (1980) found 80 percent of
impotent diabetics to have lower urinary excretion
of 17 -ketosteroids.

Several other workers have, however, failed
to confirm these findings. Hormone levels have
repeatedly been demonstrated to be within normal
limits in impotent as well as potent diabetics
(Horstmann, 1950 ; Berquist, 1954 ; Kent, 1966 ;
Eliasson et al, 1970 ; Rivarola et al, 1970 ;
Ellenberg, 1970, 1971 ; Faerman et al, 1972, 1974 ;

Fox et al, 1972 ; Kolodny et al, 1974 ; Campbell and McCulloch, 1979 and Krosnick and Podolsky, 1981). Furthermore, most of the workers have found sexual hormone therapy to be utterly ineffective in treating impotence of diabetics (Simpson,1950; Ellenberg, 1970, 1971 ; Brit. Med. J. 1974 ; Kolodny et al, 1974, 1979 ; Campbell and McCulloch, 1979 and Tattersall, 1982). In fact, these authors have found that, testosterone may be harmful as it increases the libido without improving performance - thus worsening the patients misery. Lastly, as discussed earlier, testicular biopsy and seminal fluid studies have revealed no definite changes in testicular histology, spermatogenesis, sperm count and sperm motility (Oakley, 1949 ; Keen, 1959 ; Rivarola et al, 1970 ; Ellenberg, 1970 and Faerman et al, 1972, 1974).

It seems therefore, that there is no consistent or acceptable support for an endocrinological basis for impotence occurring in diabetics. The most damaging argument is the demonstration of normal amounts of plasma testosterone (Ellenberg, 1970, 1971).

Other factors :

Diabetes renders a person more prone to a
host of other illnesses, viz., infections(particularly
balanoposthitis, urethritis and prostatitis etc.),
cardio-vascular and renal diseases which can lead to
impotence on their own because of the general catabolic
state or the physical disability engendered thereby
(Masters and Johnson, 1970 ; Kolodny et al, 1974,
1979 ; Alam et al, 1981 and Krosnick and Podolsky,
1981). Further, many of the drugs commonly prescribed
to diabetics such as antihypertensives and psychotropic
agents and alcohol can all contribute to the sexual
difficulty and must be taken into account while
assessing the relative contributions of various
aetiological factors (Kolodny et al, 1974 ; Krosnick
and Podolsky, 1981 and Tattersall, 1982).

PREMATURE EJACULATION :

Premature ejaculation is the commonest
sexual 'disturbance' in the general population
(Kinsey et al, 1948 ; Salzman, 1962 ; Masters and
Johnson, 1970 ; Adelson, 1974 ; Kaplan et al, 1974
and Levine, 1976). Kinsey et al (1948) found that

75 percent of men ejaculated within two minutes.
These authros doubted if premature ejaculation
could at all be called a disturbance. Rather they
felt that quick ejaculation might be a superior
trait - a sign of masculinity.

In view of its frequency in the population,
premature ejaculation does not appear to be a
significant problem in diabetic males. Its frequency
in the study of Kolodny et al (1974) was 2 percent
and 7.5 percent in that of Alam et al (1981).
Khandelwal et al (1981) found it to occur in 32
percent of the patients with a duration of upto
5 years as against 13 percent in those with a
duration of more than 5 years.

The cause of premature ejaculation is
predominantly psychogenic (Kolodny et al, 1979).
Organic causes such as prostatic or other genito -
urinary inflammations have been suggested but not
proved (Masters and Johnson, 1970 ; Kaplan, 1975
and Levine, 1976).

RETROGRADE EJACULATION :

Retrograde ejaculation is a condition in
which seminal fluid flows backwards into the bladder

at the time of orgasm, rather than being propelled
in a forward direction through the distal urethra.
This disorder has been reported to occur in 1-2
percent of diabetic men (Rubin and Babbott, 1958;
Keen, 1959 ; Klebanow and MacLeod, 1960; Ellenberg,
1966 ; Ellenberg and Weber, 1966 ; Ellenberg, 1971;
Kolodny et al, 1974 and Alam et al, 1981). It almost
never occurs in the general population. Khandelwal et
al (1981) found retrograde ejaculation in 20 percent
of diabetics of upto 5 years duration of diabetes.
None of the controls had it.

The cause of the problem is, almost
certainly, an autonomic neuropathy that has
progressed to the involvement of the neck of
urinary bladder (Ellenberg and Weber, 1966 ;
Bourne et al, 1971 ; Kolodny et al 1979 and
Krosnick and Podolsky, 1981). Normally the neck
of the bladder closes tightly during orgasm and
ejaculation, with the result that pressure posterior
to the prostatic urethra is so high that the seminal
fluid moves anteriorly in the direction of least
resistance. In affected diabetic men, because the
internal sphinctor of the bladder does not close
effectively there is more resistance in the forward

direction (resistance created normally by the walls of the urethra) and less resistance backwards into the bladder, since the distance is considerably shorter. Seminal fluid therefore mixes freely with urine. The diagnosis is established by finding numerous sperms in a post-coital urine specimen after having demonstrated the absence of an ejaculate or sperms in a condom used during intercourse (Kolodny et al, 1979).

Retrograde ejaculation has not received much attention and the literature dealing with it is rather scanty.

MATERIAL & METHODS

MATERIAL AND METHODS

The study was conducted in the department of Medicine, M.L.B. Medical College and Hospital, Jhansi. The sample consisted of a study group and a control group.

STUDY GROUP :

The male diabetics who attended the 'diabetic clinic' or were admitted to the medical wards of M.L.B. Medical College and Hospital, Jhansi, between March 1982 and March 1983 constituted the material for the present study. Thus the sample consisted of sixty four consecutive cases with an unequivocal evidence of diabetes. Diabetes was diagnosed according to the criteria of National Data Group of American Diabetes Association (1979) (Appendix- I).

CONTROL GROUP :

These were 50 healthy age and sex matched controls comprising of hospital personnel, medical students and attendants of the patients.

METHODS

All the patients were interviewed in an atmosphere of privacy after gaining their confidence. The history was recorded on a schedule (Appendix-II) specially prepared for the purpose.

Medical, family and personal history were obtained with special emphasis on patient's medications, alcohol intake, previous operations, injuries and history of neurologic diseases. A detailed account of family was also noted, particularly about duration of married life, number of children and the last child birth.

The patients were grouped into underweight, normal and overweight by measuring their height and weight (Appendix-III).

Diabetic status was assessed on the following lines :

1. Age of onset of diabetes.

2. Duration of diabetes.

3. Type of diabetes i.e. Type I or Type II (Cudworth, 1976, 1978).

All insulin dependent diabetics, regardless of age were included in 'type I' diabetics. Non-insulin dependent patients maintained on diet alone or with the aid of oral hypoglycaemic agents were classified as 'type II' diabetes.

4. Degree of hyperglycaemia.

The patients were categorised into three grades of severity on fasting blood sugar levels as follows (Viswanath et al, 1976) :-

(i) Mild - fasting blood sugar less than 150 mg %

(ii) Moderate - fasting blood sugar between 150 - 200 mg%

(iii) Severe - fasting blood sugar level more than 200 mg%

5. Treatment history.

6. Degree of control.

Sexual history :

Informations sought during the interrogation were as follows : onset of sexual dysfunction, progression of sexual dysfunction, interval between onset of diabetes and sexual dysfunction, current

level of sexual functioning, libido, orgasms, ejaculation (premature, absent or retrograde), morning erections, nocturnal emissions, masturbation, frequency of sexual outlets and patient's attitude towards the absence or decline in sexual performance. Wherever possible, sexual partner was also interrogated.

Impotence was considered to be present if the patient had inability to obtain or maintain an erection of sufficient firmness to permit coitus to be initiated or completed (Masters and Johnson, 1970).

The condition wherein orgasm and ejaculation persistently occurred before or immediately after penetration of the female interoitus was considered to be premature ejaculation (Schapiro, 1943).

The inability to ejaculate and experience orgasm despite the maintenance or erection was regarded as 'retarded ejaculation' (Friedman, 1973 and Kaplan, 1974) or 'ejaculatory failure' (Taylor, 1975) while orgasm without ejaculation was included in retrograde ejaculation (Andaloro and Dube, 1975).

The overall sexual functional status of the patient was categorised into normosexual, hyposexual, hypersexual and deviations (Shukla et al, 1979) (Appendix-II).

CLINICAL DATA :

After the sexual and medical histories were taken, all the patients were questioned about the presence of symptoms reported to be related to autonomic neuropathy viz., postural giddiness, nocurnal diarrhoea, disturbances of bladder sphinctor, constipation and sweating disturbances. They underwent a detailed physical examination with special reference to somatic peripheral neuropathy. Peripheral neuropathy was considered to be present if a patient complained of pain and paraesthesias and had impaiment of sensations (gloves and stockings type) with loss of ankle jerk and were finally assessed for autonomic neuropathy by the following tests :

1. Orthostatic change in pulse rate :

Pulse rate was counted first in the supine and then in the standing positions. A rise of less than 10 beats per minute on rising was considered abnormal (Nies, 1972 and Ewing et al, 1978 and 1981).

2. Orthostatic change in blood pressure :

Blood pressure in the right arm was recorded first with the patient supine and again after the patient had been standing for 10 minutes.

Orthostatic hypotension was considered to be present if there was a fall in blood pressure of at least 30/20 mm Hg with or without giddiness or syncope (Schatz et al, 1963).

3. Blood pressure response to nitroglycerine :

The blood pressure was measured in the right arm before and after sublingual administration of 0.5 mg of nitroglycerine (ANGISED), with the patient lying supine. A fall of blood pressure by more than 15 mm Hg systolic and more than 5 mm Hg diastolic was taken as an indication of autonomic neuropathy (Nies, 1972).

4. Heart rate response to Valsalva's manoeuvre :

ECG was recorded in quiet respiration and then valsalva's manoeuvre was carried out by the patient expiring against closed glottis for about 30 seconds. For this purpose nostrils were closed by hand. Again ECG was recorded and after 15 seconds pressure was abruptly relieved and ECG was recorded for further 30 seconds. Absence of tachycardia in phase II(straining maintained) suggested paralysis of the cardiac sympathetic and absence of reflex bradycardia in phase IV (after straining released) suggested paralysis of the cardiac parasympathetic (Elisberg, 1963 and Nies, 1972).

5. Residual Urine :

Patient was asked to pass urine and then a plain catheter was inserted into bladder per urethra. Amount of urine obtained after catheterisation was measured and considered as abnormal if prostatic hypertrophy was not present (Ellenberg, 1971).

A history was obtained and relevant investigations were done for vasculopathy, retinopathy and nephropathy viz., history of cerebral ischemia, intermittent claudication, myocardial ischemia, trophic ulcers, anasarca, peripheral pulsations, fundus examination, urine albumin, blood urea, ECG and other investigations wherever indicated.

Seminograms of few patients were also done.

All the patients of study group were given treatment for diabetes and whenever indicated, also for the associated psychiatric condition. Most of the patients were followed up regarding their control of diabetes and sexual functioning.

In the end, the findings were tabulated and the data analysed statistically.

OBSERVATIONS

The present work was undertaken to study the sexual functioning of male diabetics vis-a-vis that of non-diabetic males. The study group consisted of 64 consecutive male diabetics while the control group was formed by 50 age-matched ($P > 0.1$)(Table I).

TABLE I

AGE DISTRIBUTION OF THE CASES IN THE STUDY AND CONTROL GROUPS.

Age-groups (Years)	Diabetics (n=64)		Controls (n=50)	
	No.	%	No.	%
Upto 20	4	6.2	8	16.0
21 - 30	14	21.9	15	30.0
31 - 40	8	12.5	9	18.0
41 - 50	19	29.7	10	20.0
51 - 60	15	23.4	5	10.0
61 - 70	3	4.7	1	2.0
> 70	1	1.6	2	4.0
Total	64	100.0	50	100.0

$x^2 = 7.09$; d.f. = 4; $P > 0.1$

non-diabetic and apparently healthy individuals.
The two groups turned out to be similar on such
sociodemographic variables as marital status
(P $\rightarrow$ 0.05), education (P $\gt$ 0.1), occupation
(P $\gt$ 0.1) and social class (P $\rightarrow$ 0.3) (Table II
and III).

TABLE II

MARITAL STATUS OF CASES IN THE TWO GROUPS.

Marital status	Diabetics (n=64)		Controls (n =50)	
	No.	%	No.	%
Single	6	9.4	12	24.0
Married	52	81.2	36	72.0
Widower	5	7.8	2	4.0
Divorced	1	1.6	-	-

x^2 = 5.18; d.f. = 2; P $\gt$ 0.05

TABLE III

DISTRIBUTION OF CASES BY SOCIO-ECONOMIC STATUS.

	Diabetics (n = 64)		Controls (n = 50)	
	No.	%	No.	%
A. EDUCATIONAL STATUS				
Illiterate	21	32.8	25	50.0
Primary	25	39.1	16	32.0
Secondary	15	23.4	3	6.0
College/ University	3	4.7	6	12.0

$$x^2 = 3.63; \quad d.f. = 2; \quad P > 0.1$$

B. OCCUPATION				
Active	49	76.6	43	86.0
Sedentary	15	23.4	7	14.0

$$x^2 = 1.6; \quad d.f. = 1; \quad P > 0.1$$

C. SOCIAL CLASS

(Mean per capita income Rs./month)

	Diabetics		Controls	
I ($\geqslant$ 600)	2	3.1	3	6.0
II (300-599)	9	14.1	8	16.0
III (140-299)	15	23.4	16	32.0
IV (60-139)	22	34.4	11	22.0
V (< 60)	16	25.0	12	24.0

$$x^2 = 2.59; \quad d.f. = 3; \quad P > 0.3$$

As can be seen from table IV the diabetics were significantly different from controls in their weight (P $\angle$ 0.001). Nearly two thirds (62.0%) of controls were of normal weight as compared to just one-eighth (12.5%) of the diabetic group. Nearly two thirds (62.5%) of diabetics were underweight as against just one-fourth (26.0%) of controls. Similarly, overweight too was more common among diabetics (25.0%) than among controls (12.0%).

TABLE IV

DISTRIBUTION OF CASES BY WEIGHT

Weight	Diabetics (n = 64)		Controls (n = 50)	
	No.	%	No.	%
Under weight	40	62.5	13	26.0
Normal weight	8	12.5	31	62.0
Over weight	16	25.0	6	12.0

x^2 = 30.7; d.f. = 2; P $\angle$ 0.001

SEXUAL DYSFUNCTIONS :

The two groups differed significantly
with respect to their sexual functional status
(P $<$ 0.001). Nearly three-fourths (47 cases or
73.4%) of diabetics had sexual disturbances as
against just 14.0% of the controls. All but one
of the diabetics with sexual disturbances had
impotence of varying degrees. Three of them had
premature ejaculation in addition to impotence.

TABLE V

SEXUAL FUNCTIONS IN THE DIABETICS AND CONTROLS

Sexual functions	Diabetics		Controls	
	No.	%	No.	%
1. Normal	17	26.6	43	86.0
2. Impotence	46	71.9	5	10.0
- Complete(31)			(3)	
- Partial (15)			(2)	
3. Premature ejaculation	4	6.3	2	4.0
Total	67*	104.8*	50	100.0

$$x^2 = 43.47; \quad d.f. = 1; \quad P < 0.001$$

*Three cases had both impotence (Partial) and
premature ejaculation.
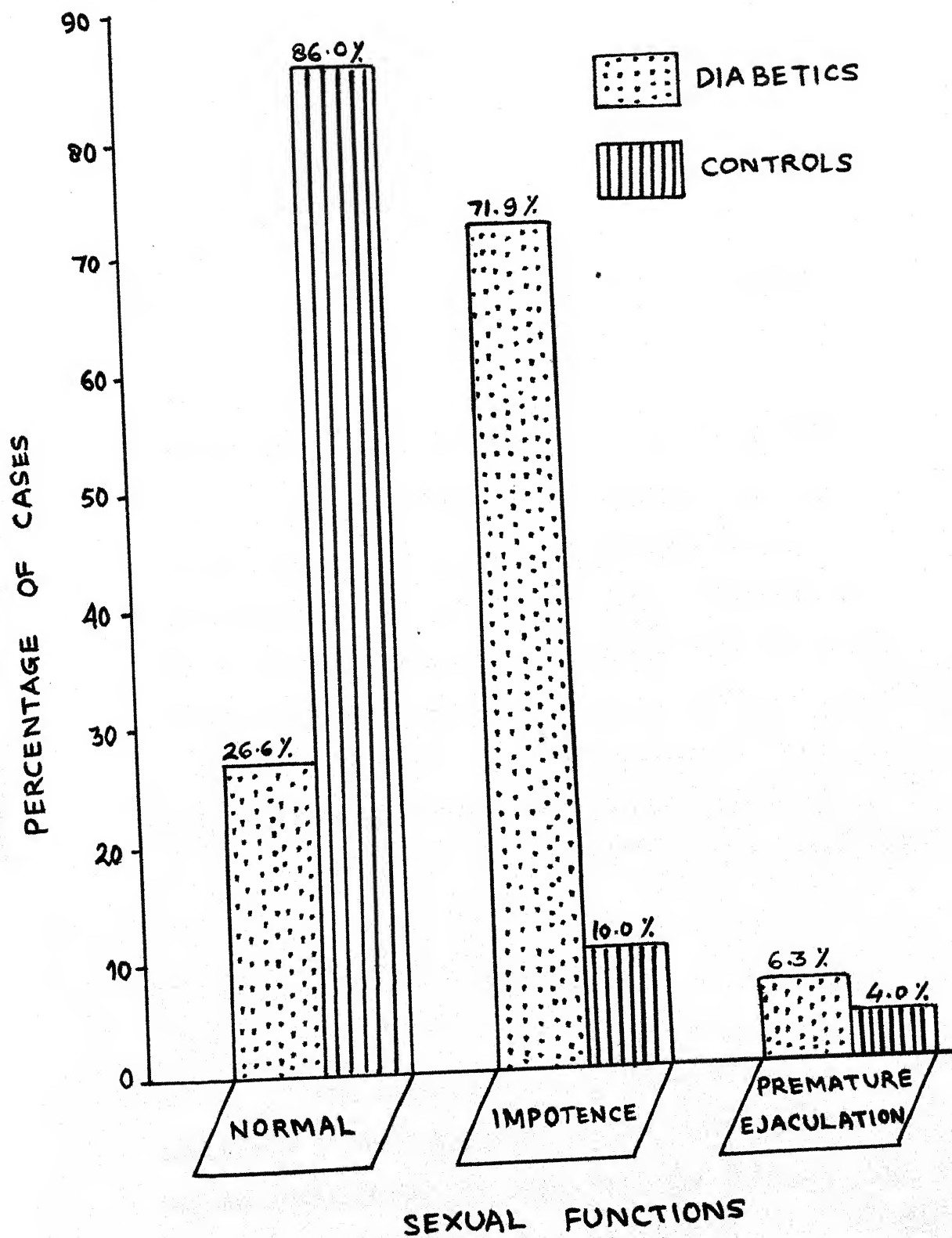
Fig. 1. SEXUAL FUNCTIONS IN DIABETICS & CONTROLS

One case had premature ejaculation as the sole
sexual disturbance. Out of 50 controls just
5 (10.0%) and 2 (4.0%) respectively, had impotence
and premature ejaculation. Of the impotent cases,
in both the groups, the dysfunction was complete
in two-thirds and partial in the remaining one-
third (Table V, Fig. 1). There was no case with
retrograde ejaculation in either of the groups.

As there was just a single case with
pure premature ejaculation, impotence could be
taken as practically the only sexual disturbance
seen in the diabetics. The description henceforth,
unless specified otherwise, would therefore pertain
to this abnormality. Various features of sexual
dysfunctions and their relation with different
aspects of diabetes will be analysed in some detail.

Features of sexual dysfunctions :

Onset :

The onset of sexual disturbances was
insidious in 45 (95.7%) of 47 affected cases of
the diabetic group. In the remaining 2 (4.3%) the
onset was acute (Table VI). As against this, in the
control group the onset was acute in 71.4% of the

cases. This difference was highly significant
(P $<$ 0.001), implying that diabetic impotence was
almost always insidious in onset while it was mostly
of acute onset in the controls.

Course :

The course of decline in sexual functions
was progressive in 91.5% of diabetics while 71.4%
of the controls had a stationary course. Only one
diabetic patient reported some improvement in
sexual functions during hospital stay (Table VI).
Thus diabetic impotence had progressive course
while stationary course was seen in controls, the
difference being highly significant (P $<$ 0.001).

Nature :

Based on the overall clinical evaluation
of the cases it was felt that the sexual dysfunction
was organic in nature in most of the diabetics (95.7%)
while it was functional in 71.4% of the controls
(Table VI). The nature of the sexual dysfunction was
thought to be organic when it was of insidious onset,
progressive course and with no history of morning
erections, nocturnal emissions, erection on
masturbation or extramarital sex.

TABLE VI

FEATURES OF SEXUAL DYSFUNCTION IN 47 AFFECTED DIABETICS

Features	No.of diabetics with sexual dysfunction (n = 47)	%
1. Onset*		
Acute	2	4.3
Insidious	45	95.7
2. Course*		
Progressive	43	91.5
Stationary	3	6.4
Regressive	1	2.1
3. Nature**		
Functional@	2	4.3
Organic@	45	95.7
4. Relation with onset of diabetes		
Antedated	4	8.5
Coincided	20	42.6
Followed	23	48.9

* In control group only 2 cases out of 7 (28.6%) had insidious onset and progressive course.

**Based on overall clinical features.

@ In control group the dysfunction was functional in 5 (71.4%) and organic in 2 (28.6%) cases.

Relation with onset of diabetes :

In most (91.5%) of the cases, sexual
dysfunctions followed (range 6 months to 8 years)
or coincided with the onset of the symptoms of
diabetes. However, in 4 (8.5%) cases the decline
in sexual functions preceded (range 3 month to
1 year) the diagnosis of diabetes (Table VI).

Frequency of sexual outlets :

Frequency of mean sexual outlets per month
in study group was less (3.2 ± 2.9) than that in the

TABLE VII

FREQUENCY OF SEXUAL OUTLETS IN STUDY AND CONTROL GROUPS

Sexual outlets* (per month)	Diabetics (n = 64)		Controls (n = 50)	
	No.	%	No.	%
Nil	37	57.8	4	8.0
1 - 4	17	26.6	18	36.0
4 - 8	3	4.7	11	22.0
8 -12	5	7.8	5	10.0
7 12	2	3.1	12	24.0
Total	64	100.0	50	100.0
Mean	3.2		6.6	
S.D.	2.9		4.9	

Statistical significant t = 4.55; d.f. = 112; P $<$ 0.001

*Approximate number at the time of study.

control group (6.6 ± 4.9). No sexual outlets
were reported by 37 (57.8%) diabetics and 4 (8.0%)
controls who were having sexual dysfunctions. The
difference was highly significant (P ∠ 0.001)
(Table VII) meaning thereby that frequency of sexual
outlets was diminished in diabetics as compared to
non-diabetics.

Libido :

An impairment in the libido or sexual
desire was seen in almost all the cases with impotence,
i.e., 45 out of 47 diabetics and 5 out of 7 controls
with sexual dysfunctions, had partial or complete
loss of libido.

Attitude of patients towards their sexual dysfunctions:

Majority of the patients of the control
group (71.4%) were worried over their sexual
dysfunctions. On the other hand, a good number of
diabetics were either indifferent 26.1%) or concerned
without being worried (41.3%) over their diminished or
absent sexual functions (Table VIII). The diabetes
itself was the main worry of these patients. Only

about a fourth (28.3%) were worried about it. Two cases (4.3%) felt happy to be relieved of the sexual desire.

TABLE VIII

ATTITUDE OF THE PATIENTS TOWARDS DECLINED/ABSENT SEXUAL FUNCTION

Attitude	Diabetics (n = 46)*		Controls (n = 7)*	
	No.	%	No.	%
Worried	13	28.3	5	71.4
Concerned but not worried	19	41.3	2	28.6
Indifferent	12	26.1	-	-
Happy	2	4.3	-	-

*Denotes number of cases with sexual dysfunction.

Relation of sexual dysfunction to age and weight
of the patients :

Age :

The frequency of impotence remained several
times higher in the diabetics, compared to controls, in
all the age groups. However, the effect of advancing
age on the frequency of impotence was much more

TABLE IX

FREQUENCY OF SEXUAL DYSFUNCTION BY AGE IN THE STUDY GROUP

Age-group (years)	Total cases	Cases with sexual dysfunction	
		No.	%
Upto 20	4	1	25.0
21 - 30	14	8	57.1
31 - 40	8	4	50.0
41 - 50	19	18	94.7
51 - 60	15	12	80.0
61 - 70	3	3	100.0
> 70	1	1	100.0
Total	64	47	73.4

$$x^2 = 12.43; \quad d.f. = 4; \quad P < 0.05$$
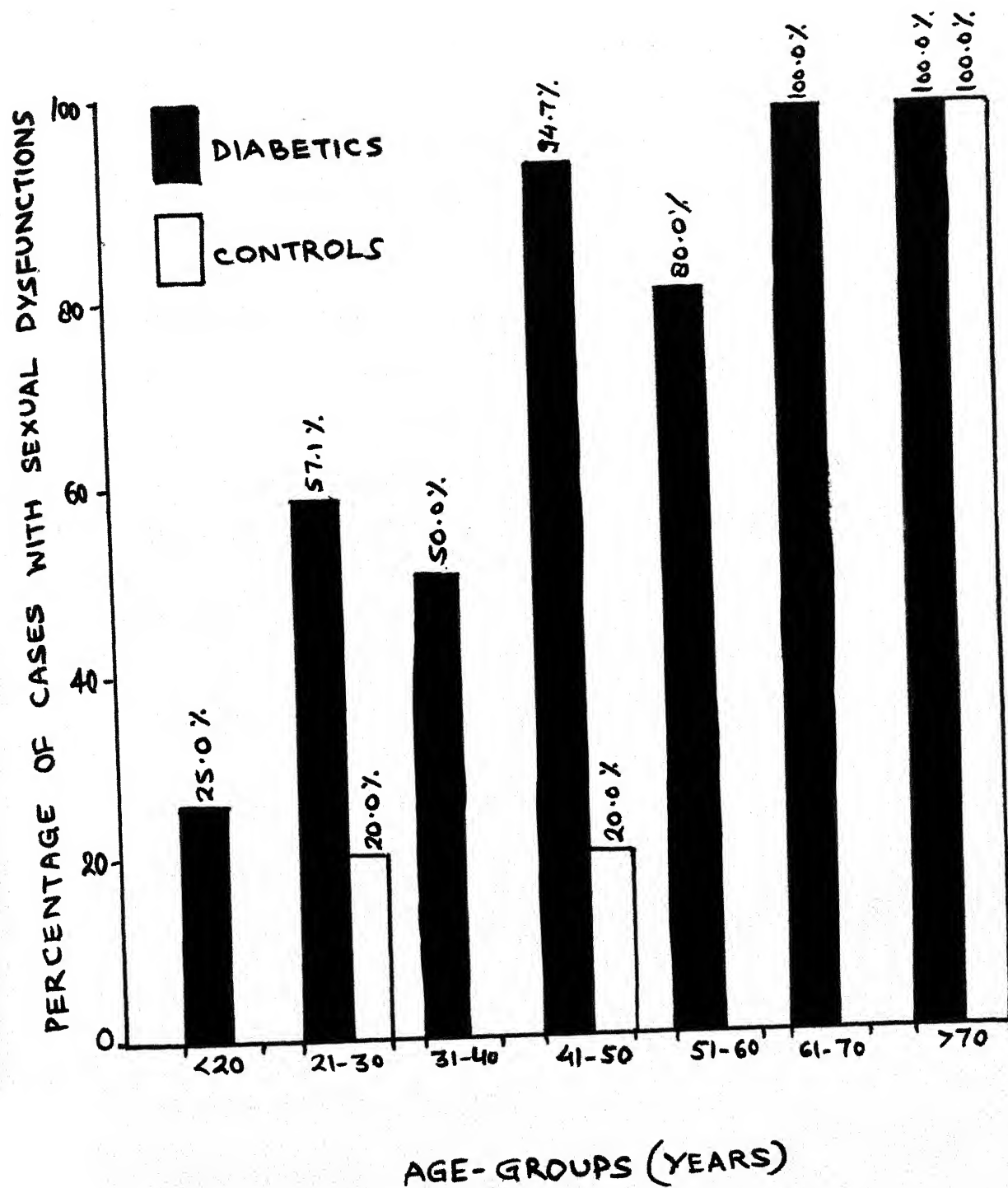
FIG. 2. AGE-WISE DISTRIBUTION OF SEXUAL DYSFUNCTIONS

pronounced in the diabetics. The frequency of impotence in the control group was 9.4% in cases upto 40 years of age and 12.5% in those beyond 40 with an absolute rise of 3.1% or a rise to the tune of 33.3% of the first value. As against this, the frequencies in the diabetics were 50% and 90% respectively, below and beyond 40 years - an absolute rise of 40% or a rise to the tune of 80% of the first value. The relation of age with the frequency of impotence was significant (P $<$ 0.05)(Table IX, Fig.2).

Weight :

Though nearly two-thirds (62.5%) of the diabetics were underweight and only a fourth (25.0%)

TABLE X

SEXUAL DYSFUNCTION BY WEIGHT IN STUDY GROUP

Weight	Total cases	Cases with sexual dysfunction	
		No.	%
Under weight	40	29	72.5
Normal weight	8	4	50.0
Over weight	16	14	87.5

$$x^2 = 3.88; \ d.f. = 2; \ P > 0.1$$

overweight, impotence was more common (87.5%) in
the latter compared to the former (72.5%) or the
patients with a normal weight (50.0%). However, there
was no significant effect of weight on the sexual
functions (P $>$ 0.1), proving that diabetes and
not the weight was related to impotence (Table X).

Sexual dysfunction in relation to various aspects
of diabetes :

Diabetic status of the study group and
relationship of different variables of diabetes with
sexual function has been shown in table XI. There was
a preponderance of type I diabetics (70.3%), symptomatic
(93.7%), uncontrolled (89.1%) and those with one or more
diabetic complications (87.5%). As regards the age of
onset of diabetes nearly half of the cases were below
40 years of age. At the time of consultation 43.8%
cases were taking insulin while 53.1% were on oral
hypoglycaemics. However, majority of the patients
were on irregular treatment.

The prevalence of sexual dysfunction was
much more in symptomatic (76.7%) diabetics ; in
those above 40 years of age (87.1%); and in those
who were on oral hypoglycaemics (88.2%). These
findings were statistically significant (P $<$ 0.05).

TABLE XI

DIABETIC STATUS OF THE STUDY GROUP

Variables	Total cases (m=64)	Cases with sexual dysfunction (n=47)		Statistical significance d.f.=1	
		No.	%	x^2	P
1. Type of diabetes					
Type I	45	32	71.1	0.42	>0.5
Type II	19	15	78.9		
2. Symptomatology					
Asymptomatic	4	1	25.0	5.13	<0.05
Symptomatic	60	46	76.7		
3. Age of onset					
<40 years	33	20	60.6	5.75	<0.05
>40 years	31	27	87.1		
4. Past treatment*					
Insulin	28	16	57.1	7.75	<0.01
Oral hypoglycaemics	34	30	88.2		
5. Control of diabetes					
Controlled	7	5	71.4	0.016	>0.8
Uncontrolled	57	42	73.7		
6. Diabetic complications					
Present	56	42	75.0	0.56	>0.3
Absent	8	5	62.5		

*8 cases were not taking any treatment and 5 were
taking both insulin and oral drugs.

However, there was no significant relation between
the frequency of sexual dysfunctions and the type
of disease (Type I or II) (P $\nearrow$ 0.5), its control
(P $\nearrow$ 0.8) or its complications (P $\nearrow$ 0.3).

Duration :

Mean duration of diabetes was 4.3 $\pm$ 3.4
years (range 0-20 years). The patients with sexual
dysfunction had a diabetes of somewhat longer
duration (4.6 $\pm$ 3.5 years) than those with normal
sexual functions (3.6 $\pm$ 3.2 years). However, the

TABLE XII

SEXUAL DYSFUNCTION IN RELATION TO DURATION OF DIABETES

Duration of diabetes (years)	Total cases (n=64)	Cases with sexual dysfunction	
		No.	%
$\angle$ 1	12	8	66.7
1- 5	33	23	69.7
6-10	15	13	86.7
$\nearrow$ 10	4	4	75.0

$$x^2 = 1.67; \text{ d.f. } = 3; \text{ P} \nearrow 0.5$$

difference failed to reach the level of statistical
significance (P > 0.5) (Table XII). Thus there was
no relation between the frequency of sexual
dysfunctions and the duration of diabetes.

Severity :

Nearly two-thirds (43 or 67.2%) of cases
were having a severe degree of hyperglycaemia (Table
XIII). In all the three groups, i.e., mild, moderate

TABLE XIII

SEXUAL DYSFUNCTION IN RELATION TO SEVERITY OF DIABETES

Severity (Fasting blood sugar level)	Total cases (n=64)	Cases with sexual dysfunction (n=47)	
		No.	%
Mild (FBS < 150 mg%	7	5	71.4
Moderate (FBS 150-200 mg%)	14	10	71.4
Severe (FBS > 200 mg %)	43	32	74.4

$$x^2 = 0.065; \quad d.f. = 2; \quad P > 0.95$$

TABLE XIV

ASSOCIATION BETWEEN DURATION AND SEVERITY OF DIABETES

Duration (years)	Total cases (n=64)	Severity					
		FBS $\angle$ 150 mg%		FBS 150-200mg%		FBS $\diagup$ 200mg%	
		No.	%	No.	%	No.	%
$\angle$ 1	12	2	16.7	3	25.0	7	58.3
1 - 5	33	3	9.1	7	21.2	23	69.7
6 -10	15	-	-	4	26.7	11	73.3
$\diagup$ 10	4	2	50.0	-	-	2	50.0

$$x^2 = 0.024; \quad d.f. = 2; \quad P \diagup 0.975$$

and severe, the occurrence of sexual dysfunctions was more or less same (71.4%, 71.4% and 74.4% respectively in three groups), there being thus no relation between the severity of diabetes and frequency of sexual disturbances (P $\diagup$ 0.95). Further, there was no association between duration and severity of diabetes (P $\diagup$ 0.975) (Table XIV).

Mode of treatment :

Though the patients on insulin treatment (35 or 54.7%) were much more in number than those on oral hypoglycaemics (19 or 29.7%), the sexual dysfunctions were more frequent (78.9%) in patients on oral hypoglycaemics. Further, all the cases who were taking both insulin and oral drugs simultaneously had sexual disturbances. Nevertheless, the difference was not statistically significant (P $\nearrow$ 0.05)(Table XV).

TABLE XV

SEXUAL DYSFUNCTION IN RELATION TO MODE OF TREATMENT OF DIABETES

Treatment of diabetes*	Total cases (n=64)	Cases with sexual dysfunction (n=47)	
		No.	%
Insulin	35	22	62.9
Oral hypoglycaemics	19	15	78.9
Insulin + Oral hypoglycaemics	10	10	100.0

$$x^2 = 5.93; \quad d.f. = 2; \quad P \nearrow 0.05$$

*No cases were observed on dietary treatment only.

Complications :

Out of 51 (79.7%) diabetics with neurological complications 40 (78.4%) had sexual dysfunctions and out of 20 (31.3%) cases with vascular complications 14 (70.0%) had sexual dysfunctions. There rates were considerably higher than those in patients without complications (62.5%) (Table XVI, Fig. 3).

TABLE XVI

SEXUAL DYSFUNCTION IN RELATION TO COMPLICATIONS OF DIABETES

Complications*	Total cases (n=64)		Cases with sexual dysfunction (n=47)	
	No.	%	No.	%
(i) Nil	8	12.5	5	62.5
(ii) Neurological	51	79.7	40	78.4
Peripheral neuropathy 42 (65.6)			36 (85.7)	
Autonomic neuropathy 36 (56.2)			31 (86.1)	
(iii) Vascular	20	31.3	14	70.0
Microangiopathy 16 (25.0)			12 (75.0)	
Macroangiopathy 10 (15.6)			8 (80.0)	

*Some patients had multiple complications.
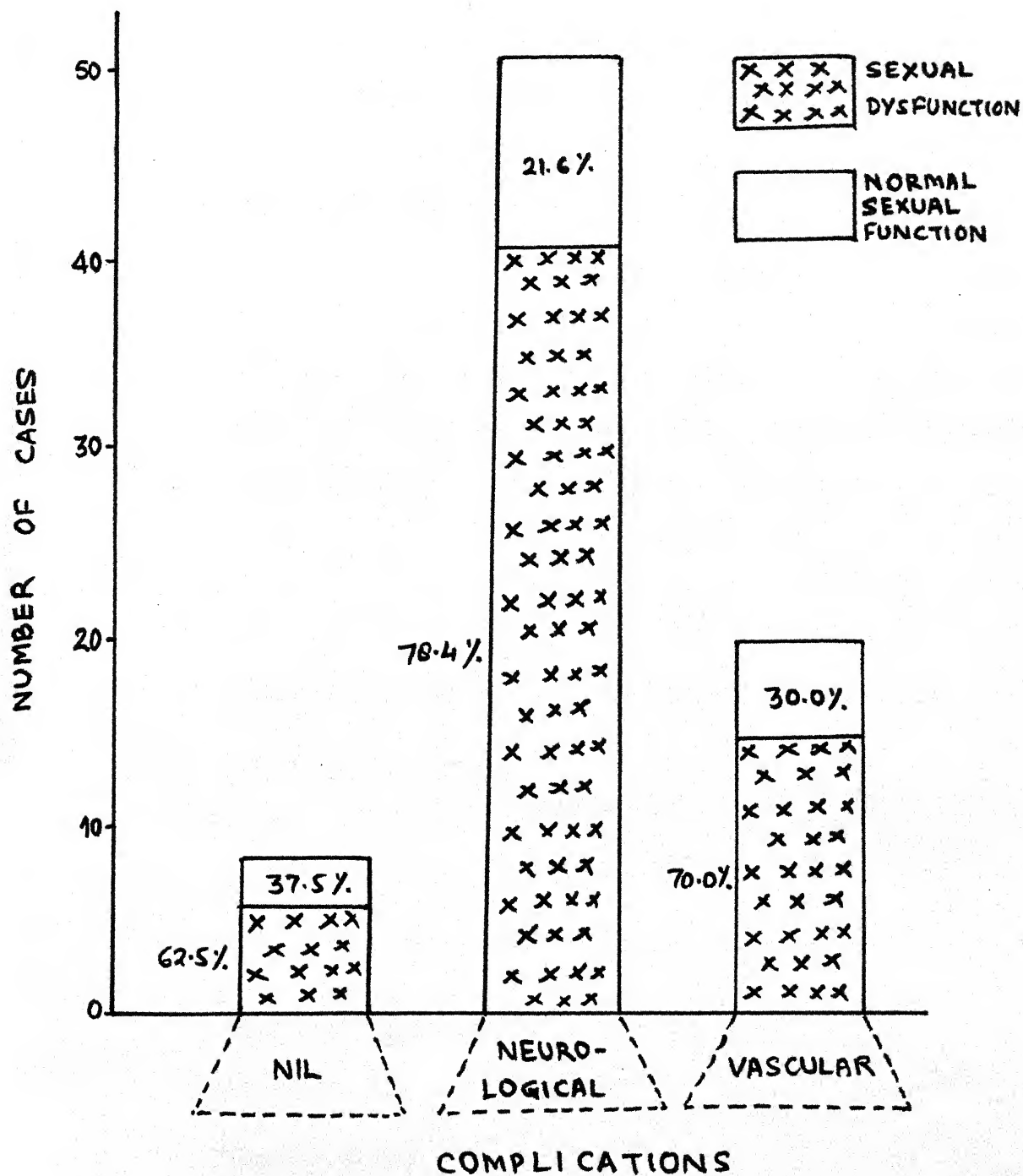
FIG. 3. FREQUENCY OF SEXUAL DYSFUNCTION IN RELATION TO COMPLICATIONS OF DIABETES

Of the 51 cases with neuropathy, peripheral neuropathy was present in 42 cases (65.6%) and autonomic neuropathy in 36 (56.2%), there being 29 (69%) cases in whom both types of neuropathies were present. Sexual disturbances were associated with equal frequency (86.0%) with these two types of neuropathy. Similarly, the frequency of sexual disturbances was almost same in cases with micro- (75.0%) and macroangiopathy (80.0%). Neurological and vascular complications appeared to be independent of each other (P $\mathbf{7}$ 0.1) (Table XVII).

TABLE XVII

ASSOCIATION BETWEEN VASCULOPATHY AND NEUROPATHY

	Total cases (n=64)	Neuropathy present (n=51)	
		No.	%
Vasculopathy present	20	18	90.0
Vasculopathy absent	44	33	75.0

$$x^2 = 1.91; \quad d.f. = 1; \quad P \; \mathbf{7} \; 0.1$$

Thirty six (56.2%) cases had evidence of autonomic neuropathy. The frequency of sexual dysfunctions was significantly higher (86.1%) in cases with autonomic neuropathy compared to that in cases without it (57.1%) (P $\angle$ 0.01) (Table XVIII).

TABLE XVIII

CORRELATION OF SEXUAL DYSFUNCTION WITH NEUROPATHIES

Neuropathy	Total cases (n=64)	Diabetics with sexual dysfunction (n=47)	
		No.	%
(i) Peripheral			
Present	42	36	85.7
Absent	22	11	50.0

x^2 = 9.44; d.f. = 1; P $\angle$ 0.01

(ii) Autonomic			
Present	36	31	86.1
Absent	28	16	57.1

x^2 = 6.77; d.f. = 1; P $\angle$ 0.01
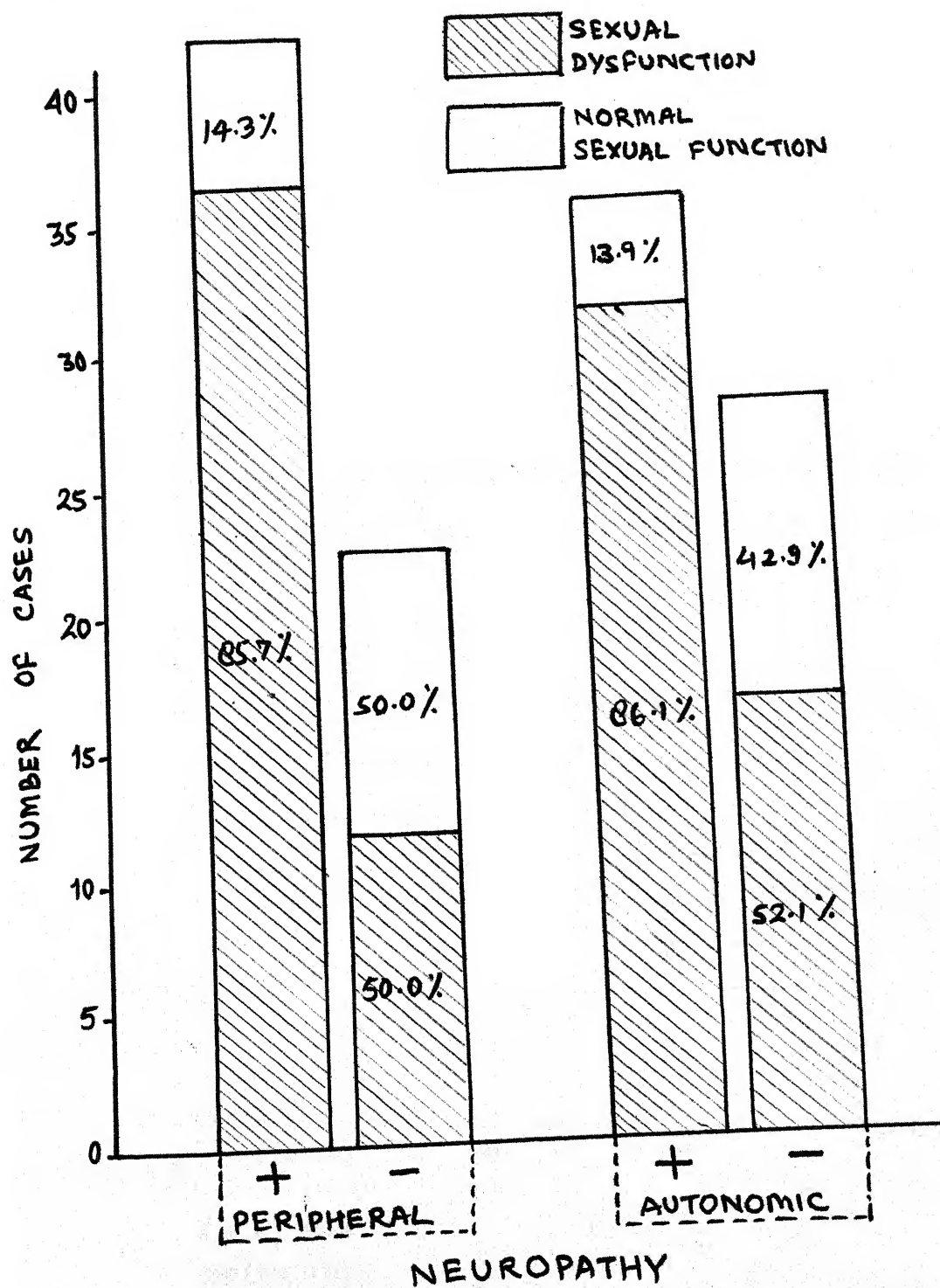
Fig. 4. FREQUENCY OF SEXUAL DYSFUNCTION IN RELATION TO NEUROPATHIES

A total of 42 (65.6%) patients had evidence of peripheral neuropathy as manifested by pain, paraesthesias, absent ankle jerk and impaired sensations. Of these 85.7% had sexual dysfunctions - a frequency significantly higher than that (50.0%) recorded in cases without peripheral neuropathy (P $\angle$ 0.01)(Table XVIII,Fig 4).

As can be seen from the table XIX, out of 42 cases with peripheral neuropathy 29 (69.0%)

TABLE XIX

ASSOCIATION BETWEEN PERIPHERAL AND AUTONOMIC NEUROPATHIES

	Total cases (n=64)	Cases with autonomic neuropathy	
		No.	%
Peripheral neuropathy present	42	29	69.0
Peripheral neuropathy absent	22	7	31.8
Total	64	36	56.3

x^2 = 8.13; d.f. = 1; P $\angle$ 0.01

had autonomic neuropathy as well, the two being
thus closely associated (P $<$ 0.01). The occurrence
of neuropathy had no relation with the age of
patients (P $>$ 0.1) (Table XX). However, there was
a definite relation with duration of diabetes,

TABLE XX

ASSOCIATION BETWEEN AGE AND NEUROPATHY

Age-group (years)	Total cases (n=64)	Cases with neuropathy (n = 51)	
		No.	%
Upto 20	4	3	75.0
21- 30	14	9	64.3
31- 40	8	7	87.5
41- 50	19	18	94.7
51- 60	15	10	66.7
61- 70	3	3	100.0
$>$ 70	1	1	100.0

$$x^2 = 5.33; \quad d.f. = 3; \quad P > 0.1$$

the frequency getting higher with increasing
duration (P $\angle$ 0.05) (Table XXI). No relation
was observed between severity of hyperglycaemia
and incidence of neuropathy (P $\daleth$ 0.25) (Table
XXII).

TABLE XXI

ASSOCIATION BETWEEN DURATION OF DIABETES AND INCIDENCE OF NEUROPATHY

Duration (years)	Total cases (n=64)	Cases with neuropathy (n=51)	
		No.	%
$\angle$ 1	12	7	58.3
1 - 5	33	26	78.8
6 -10	15	15	100.0
$\daleth$ 10	4	3	75.0

$$x^2 = 6.25; \ d.f. = 2; \ P \angle 0.05$$

TABLE XXII

ASSOCIATION BETWEEN SEVERITY OF DIABETES AND
NEUROPATHY

Severity (Fasting blood sugar level)	Total cases (n=64)	Cases with neuropathy (n=51)	
		No.	%
Mild (FBS $<$ 150 mg%)	7	6	85.7
Moderate (FBS 150-200 mg%)	14	9	64.3
Severe (FBS $>$ 200 mg%)	43	36	83.7

$$x^2 = 2.72; \quad d.f. = 2; \quad P > 0.25$$

PSYCHIATRIC STATUS :

Out of all the patients of study and control groups, only 2 patients from the control group had some psychiatric problem. One was diagnosed as a case of anxiety neurosis while the other was having endogenous depression. Both of these patients had premature ejaculation.

FOLLOW-UP :

Only 26 cases of study group came for follow-up, out of which 24 were with sexual dysfunctions. Diabetes of 15 (62.5%) cases was under control while 9 (37.5%) cases took irregular treatment after discharge from the hospital and consequently remained uncontrolled. Sexual status remained unchanged in all but one who reported about 10% improvement in his erectile ability after three months antidiabetic treatment (Table XXIII).

TABLE XXIII

FOLLOW-UP OF CASES*

Diabetic status	Diabetics with sexual dysfunction				Diabetics without sexual dysfunction		
	Cases follow-ed-up	Improved follow-up	Station-ary	Worsened	Cases follow-ed-up	Normal through-out	Any impairment
Controlled	15 (62.5%)	1 (6.7%)	14 (93.3%)	-	-	-	-
Uncontrolled	9 (37.5%)	-	9 (100.0%)	-	2 (100.0%)	2 (100.0%)	-
Total	24 (92.3%)	1 (4.2%)	23 (95.8%)	-	2 (7.7%)	2 (100.0%)	-

*only 26 cases came for follow-up.

DISCUSSION

Diabetes mellitus is a fairly common
disease all over the world. In India its prevalence
is around 3 percent (Berry et al, 1966; Patel et al,
1966; Satyanarayan et al, 1966; Gupta et al, 1970;
Tripathy, 1971 and Ahuja et al, 1972). At this
rate there would be nearly 20 million diabetics
in the country. Being chronic and of obscure
aetiology, the disease poses numerous tantalising
problems in its understanding and management-both
for the patient and his physician. Sexuality is
one such poorly understood facet of diabetes.
Though perceptive clinicians have been noticing
and commenting upon the impaired sexual functioning
of the diabetic since the turn of the century (Von
Noorden, 1903 and Naunyn, 1906), the subject has
received little systematic scientific attention.
This is particularly so in our country (Alam et al,
1981 and Gahlaut and Sharma, 1982), where we did
not have prevalence rates of sexual disturbances
even for the general population (Nakra et al, 1977).

In the light of the above, it is hardly
surprising that opinions have varied widely. Thus
while on the one extreme, Joslin (1959) felt sexual
disturbances to be rare in diabetics, Khandelwal
et al (1981) found them in as many as 84 percent
of their patients. Most of the workers have reported
figures in between these two extremes, i.e., around
fifty percent of male diabetics have been found to
have sexual disturbances (Rundles, 1945; Rubin and
Babbott, 1958; Schoffling et al, 1963; Kolodny et
al, 1974; Herman et al, 1978 and Krosnick and
Podolsky, (1981). The variations in the reported
frequency of sexual disturbances in the diabetics
is most probably a reflexion of the differing case
selection criteria and varying intensity of the
interrogations. Thus while some workers have
investigated a relatively small sample of cases
rather thoroughly (Lester et al, 1980 and Fairburn
et al, 1982), others have studied a large case
material rather superficially (Ellenberg, 1971;
Combbell and McCulloch, 1979 and Alam et al, 1981).
Not only that, authors have often used the terms
libido, potency and fertility rather vaguely,
indiscriminately and interchangeably, thus
creating considerable confusion. It is essential
that these terms are used in their precise and

accurate contexts since these functions can be affected individually. Further, barring a few, the studies on the sexual functions of diabetics have been uncontrolled, vitiating any valid conclusions regarding the frequency of sexual disturbances in diabetics vis-a-vis that in non-diabetics.

To obviate these problems, in the present study, a reasonably adequate number (64) of consecutive diabetics, irrespective of duration, severity and age of onset, fulfilling strict-diagnostic criteria were interrogated intensively and investigated thoroughly to arrive at an overall assessment of their sexual functioning. Fifty age matched, non-diabetics and apparently healthy individuals were subjected to identical interrogations to serve as controls. The two groups turned out to be comparable in marital and socio-economic variables as well. The interrogation, besides covering various aspects of diabetes and sexuality, incorporated specific questions to unearth any existing psychopathology which could account for the sexual dysfunction. The cases were treated appropriately for diabetes and were followed up at suitable intervals so as to elucidate the

effect of control or otherwise of diabetes upon
the progress of sexual disturbances.

In the present study nearly three fourths
(73.4%) of the diabetics had sexual disturbances as
compared to just 14.0% of the controls, the difference
being highly significant (P $\angle$ 0.001). This figure
was considerably higher than those reported by most
of the earlier workers viz., 10% by Gahlaut and
Sharma (1982); around 25% by Lester et al (1980)
and Noronha et al (1981); around 35% by Alam et al
(1981) and Campbell and McCulloch (1979) and around
50% by Rundles (1945), Rubin and Babbott (1958),
Frank et al (1978) and Gebhard and Johnson (1979).
The present high figures could be because of the
intensity and thoroughness of the interrogations,
employed in the present study. As Tattersall (1982)
has pointed out, patients do not divulge their
sexual problems unless asked for specifically by
the physician in an atmosphere of privacy. This
could be the reason for Joslin's (1959) comment
that "sexual disturbances were exceedingly rare in
diabetics". On the other hand even higher figures
(84 percent) than ours (73.4 percent) have been
reported by Khandelwal et al (1981).

There have been very few controlled
studies and their findings at wide variance. Most
of the workers have compared their results with

the frequency of sexual disturbances for the
American population by Kinsey et al (1948) or
by Masters and Johnson (1970). This way, the
consensus of opinion is that sexual disturbances
are about five times as common amongst diabetics
as in the general population (Rubin and Babbott,
1958; Frank et al, 1978 and Gebhard and Johnson,
1979). The figures of the present study are in
absolute agreement, 73.4% and 14.0% respecitvely
of the diabetics and controls having sexual
disturbances-a ratio of slightly above 5 : 1. The
foregoing discussion makes it glaringly clear that
sexual problems of diabetics are enormous, more
than half of them having one disturbance or the
other. At this rate out of nearly 20 million
estimated diabetics in the country, more than 10
million would be plagued by distressing sexual
problems - a staggering figure indeed!

Out of the 47 cases (73.4%) with sexual
disturbances all but one had impotence (erectile
failure) of varying degrees. Three of them had
premature ejaculation in addition to impotence. Only
one case had premature ejaculation as the sole sexual
disturbance. There was no case with retrograde
ejaculation. Thus impotence could be taken as the

prototype of the sexual disturbance seen in the diabetic patients. The discussion henceforth will dwell largely upon its various aspects. The other two disturbances viz., premature- and retrograde ejaculation will be discussed briefly thereafter.

IMPOTENCE :

The onset of impotence was insidious in most (95.7%) of the cases in the study group and the course progressive in nearly ninety percent - only one patient reporting improvement, that too very minimal, in sexual functions with treatment of diabetes. The overall clinical evaluation favoured the opinion that the sexual dysfunction was organic in nature in most of the diabetics (95.7%) because there was absence or marked reduction in the frequency of morning erections, nocturnal emissions or erections on masturbation. In most of the cases (91.5%) sexual dysfunction followed (range of interval being 6 months to 8 years) or coincided with the onset of the symptoms of diabetes. However, in 4 (8.5%) cases the decline in the sexual functions preceded (range of interval being 3 months to 1 year) the detection of diabetes. Diabetics, in general, had significantly lowered frequency of sexual outlets compared to the control group. Thirty seven (57.8%) diabetics did not have any outlets eversince the onset of their sexual

dysfunction. All but two patients with impotence
had significant lowering of sexual desire. The
impotence was complete in two thirds (31 of 46 cases
with the dysfunction) of the cases and partial in
the remaining one third.

The features of the cases in the present
series do not completely fit into any of the two
types of diabetes-related impotence that have
classically been described (Kolodny et al, 1979).
In the first, failure of erection has been described
to occur in the context of poor diabetic control, is
accompanied by loss of interest in sex and general
malaise, and the symptoms reverse with the control
of diabetes (Oakley, 1949; Keen, 1959 and Kolodny
et al, 1979). Our cases did not conform to this
type since there was no relation between the sexual
dysfunction and the mode or degree of control of
diabetes. The second type, said to be characteristic
of diabetes, has been described as progressive and
irreversible in which all erections are affected
and which is associated with no loss of sexual
interest. The cases in the present series did not
entirely fit into this category either, since they
had marked loss of sexual desire and since the
impotence was partial in a substantial proportion
(one-third) of the cases, i.e., the patients had
erections though there were rather less frequent
or of less intensity.

Thus our cases had rather "intermediate features". Similar findings have been reported by Schiavi and Hogan (1979) and Scott et al (1980). The validity of the clinical stereotypes of the diabetic impotence, referred to above have recently been questioned by other workers too (Fairburn et al, 1981, 1982 and Tattersall, 1982). Fairburn et al (1982) examined a small number (27) of patients in detail and found that morning/spontaneous erections were present at a reduced frequency and intensity in most of the diabetics with impotence. They therefore questioned the "either or" concept of the psychogenic-organic diamension and emphasised that these two factors may interact and therefore it should be not surprising that there are no simple associations. The erectile failure of diabetics is mostly the result of a progressive physical disorder and therefore "organic" in nature. Superimposed upon this is the psychological reaction of the patient and his partner which may significantly worsen the problem-the "Psychogenic" or the "functional" component of the problem. Viewed this way it may be helpful to improve the gloomy prognosis of diabetic impotence by appropriate counselling to minimise the psychological reaction of the patient to sexual dysfunction (Renshaw, 1975, 1979; Fairburn et al,

1982 and Tattersall, 1982). These authors concluded that the current view of diabetic impotence was misleadingly oversimplified. Pure "organic" or pure "psychogenic" impotence is an exception with most of the cases being characterised by a complex mixture of organic and psychosomatic features.

Our finding that there was a marked impairment of libido (sexual desire) in 45 out of 47 diabetics with impotence was in sharp contrast to the observations of most of the other workers (Rubin and Babbott, 1958; Ellenberg, 1971; Kolodny et al, 1974; Lester et al, 1980; Moses et al, 1980 and Kronmick and Podolsky, 1981). However, there are few reports conforming to our findings that libido does show a decline (Alam et al, 1981; Khandelwal et al, 1981 and Fairburn et al, 1982). Alam et al (1981) reported impairment of libido in 70 percent of their cases and Fairburn et al in 44 percent. Unfortunately, there being no objective measurement of sexual interest, data are subjective and anecdotal.

The frequency of impotence was several times higher in the diabetics, compared to controls, in all age groups. However, the frequency beyond 40 years compared to that upto 40 years showed an

80% rise as against just 33.3% in the controls. These findings of ours were in agreement with those of Schoffling et al (1963) who reported 29 percent frequency of impotence in diabetics under the age of 30 years as against 73 percent beyond 60 years. Similarly, Alam et al (1981) reported maximum incidence in the fifth decade. Thus although sexual vigour declines with age even in the normal population (Kinsey et al, 1948), in the diabetics this relation becomes very much exaggerated (Rubin and Babbott, 1958; Klebanow and MacLeod, 1960; Schoffling et al, 1963; Kolodny et al, 1974; Marmor, 1975; Alam et al, 1981 and Gahlaut and Sharma, 1982).

Similar to the observations of Ellenberg (1971), Kolodny et al (1974) and Moses et al (1980) we found no relation between the frequency of impotence and the duration of diabetes. Rubin and Babbott (1958) found 70 percent incidence of impotence in diabetics with upto one year duration as against 47 percent in those with a duration of of 1-5 years. On the other hand, Khandelwal et al (1981) and Gahlaut and Sharma (1982) found the incidence of impotence to be proportional to the duration of diabetes.

We did not find any relation between
the frequency of impotence on the one hand and
severity or mode/degree of control of diabetes on
the other. Gahlaut and Sharma (1982) reported a
significant relation between impotence and severity
of diabetes. This relationship has, however, not
been confirmed by others (Rubin and Babbott, 1958;
Ellenberg, 1971 and Khandelwal et al, 1981).
Similarly, lack of the effect of the mode/degree
of control of diabetes has been observed by Kolodny
et al (1974) and Marmor (1975). Only one patient,
in the present study showed some improvement, that
too very little, with the control of diabetes. Our
findings thus support the view held by many that
diabetic impotence is, by and large, irreversible.
Some workers have, however, reported improvement
with anti-diabetic treatment, in the impotence
occurring early in the course of diabetes. This
they have explained on the ground that in such
cases the impotence was a reflexion of the
associated malnutrition, general malaise and
metabolic imbalance (Oakley, 1949; Rubin and
Babbott, 1958; Ellenberg, 1971; Kolodny et al,
1974; Hastings, 1975 and Moses et al, 1980).

It has been held for long that angiopathy
and neuropathy in diabetics go hand in hand (Rundles,
1945). However, subsequent workers have failed to
confirm this relation (Dolman, 1963 and Vijayan
et al, 1971). In the present study too, there was
no definite relation between these two complications
of diabetes. Thus it appears that neuropathy and
angiopathy are mutually independent associations
of the disease.

The reported incidence of neuropathy in
diabetics varies greatly, figures ranging from 4 to
93 percent. In the present study, its frequency was
79.7 percent. This great variation has been mainly
due to the differences in the criteria used for
the diagnosis of neuropathy. In the present study,
subjective symptoms and objective signs were taken
into consideration for evaluation of neuropathy.
This seems to be the best method for the purpose
in the clinical practice.

Earlier workers believed that neuropathy
in the diabetics was very much related to age of
the patients, being more common in the elderly
diabetics (Rundles, 1945; Martin, 1953 and Joslin,
1959). In the present study, like that of Ellenberg
(1970), no such relation could be established.

However, we found the frequency of neuropathy to rise steadily with the increasing duration of diabetes. Similar correlation has been recorded by Patel et al (1966) and Vijayan et al (1971). On the contrary, Bhu and Bharadwaj (1959) did not find any such correlation. The positive relation between the duration of diabetes and neuropathy is difficult to account for. It would not be legitimate to think that neuropathy gradually progresses for the time of onset of diabetes, as it is well known that many a time patients present initially with neurological complications (Ellenberg, 1970). Therefore the correlation observed between the duration of diabetes and neuropathy seems to be just circumstantial.

A higher incidence of neuropathy with increasing severity has been observed by Vijayan et al (1971). In the present study no such correlation could be established (Table XXII) - an observation which is in line with those of Locke (1953) and Ellenberg (1970).

In the present series, peripheral neuropathy was present in 65.6 percent of diabetics and autonomic neuropathy in 56.2 percent, there being a strong association between the two types. Sexual disturbances were present in around 86 percent of the cases with evidence of neuropathy of either type. Noronha et al

(1981) found autonomic neuropathy in 87.8 percent
of diabetics. However, they included impotence as
one of the features of autonomic neuropathy.
Ellenberg's (1971) work presents careful
documentations of the importance of neuropathy in
the pathogenesis of impotence in diabetic males.
Going by clinical examination alone, we detected
neuropathy in significantly greater proportion of
cases with impotence than in the potent diabetics,
thus supporting the contention that impotence in
diabetes was most likely to be due to neurogenic
factors.

PREMATURE EJACULATION :

Only one case in the study group
(diabetics) had pure premature ejaculation. There
were further three cases who had premature
ejaculation in addition to partial impotence. Thus
a total of 4 (6.3%) of the diabetics had the
disturbance. In view of its frequency, premature
ejaculation does not therefore appear to be a
significant problem in the diabetic males since
lower frequencies have been reported by other
workers also, e.g., 2 percent by Kolodny et al
(1974) and 7.5 percent by Alam et al (1981).

The disturbance is rather overshadowed by the over-riding frequency of erectile failure.

The frequency of premature ejaculation in the general population has been reported to be very much higher (Kinsey et al, 1948; Salzman, 1982; Masters and Johnson, 1970; Adelson, 1974; Kaplan et al, 1974 and Levine, 1976). However, only two cases (4.0%) of the controls in the present study had the disturbances. Its cause is mainly psychogenic (Kolodny et al, 1979) and since our controls were selected from among apparently healthy individuals, the prevalence of premature ejaculation could also be expected to be low. Both the cases with the disturbance, in the control group, were having psychiatric problems; one had anxiety neurosis and the other had endogenous depression. Nakra et al (1977) found high frequency (25.3 percent) of premature ejaculation in cases selected from those attending a psychiatric clinic which further attests to its psychogenic origin.

RETROGRADE EJACULATION :

This disorder has been reported to occur in 1-2 percent of diabetics (Rubin and Babbott,

1958; Keen, 1959; Klebanow and MacLeod, 1960;
Ellenberg, 1971; Ellenberg and Weber, 1966;
Kolodny et al, 1974 and Alam et al, 1981). It
almost never occurs in the general population.
There was no such case in either of the two groups
in the present study. This could be because we
studied only 64 diabetics - a number rather small
for studying the frequency of a relatively uncommon
disturbance like retrograde ejaculation. The
disorder is almost certainly organic in origin,
most probably due to autonomic neuropathy that
has progressed to involve the neck of the urinary
bladder (Ellenberg and Weber, 1966; Bourne et al,
1971; Kolodny et al, 1979 and Krosnick and Podolsky,
1981).

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

The present work was undertaken to study the sexual functioning of male diabetics vis-a-vis that of non-diabetic males with the objectives to find out the prevalence, pattern and probable aetiological factors of sexual disturbances and to correlate such disturbances with various aspects of diabetes.

Sixty four consecutive cases with an unequivocal evidence of diabetes attending the diabetic clinic or admitted to the medical wards of M.L.B.Medical College and Hospital, Jhansi and fifty age-matched apparently healthy controls were interviewed in an atmosphere of privacy. A detailed history covering the medical, family and personal aspects was obtained and the diabetic status was assessed with special reference to the age of onset, duration, type, severity, treatment and control of diabetes.

Sexual history was recorded in detail. If any disturbance was present, details of its onset, progress, nature and type were enquired

into. All the patients were thoroughly examined and investigated for the complications of diabetes viz, neuropathy - autonomic and peripheral, vasculopathy and nephropathy. Different variables of diabetes were studied in relation to sexual status of the patients and simultaneously sexual functions were also compared with those of non-diabetics.

The following conclusions could be drawn from the present study :

(i)   The prevalence of sexual disturbances in diabetics was very high (73.4%) as compared to non-diabetics (14.0%), the difference being statistically highly significant (P $\angle$ 0.001).

(ii)   All but one of the 47 diabetics having sexual disturbances were impotent. Premature ejaculation was the sole disturbance in only one case. No case had retrograde ejaculation.

(iii)   The nature of sexual dysfunction was organic in 95.7 percent diabetics while it was functional in 71.4 percent of non-diabetics.

(iv)   Mean frequency of sexual outlets was much less in diabetics (3.2 $\pm$ 2.9 per month), then

that in the control group (6.6 ± 4.9 per month), the difference being significant statistically (P $<$ 0.001).

(v)   Almost all impotent cases, either diabetic (45 out of 47) or non-diabetic (5 out of 7), had partial or complete loss of libido.

(vi)   The frequencies of impotence in the diabetics were 50 percent and 90 percent respectively, below and beyond 40 years of age. Thus the relation of age with the frequency of impotence was significant (P $<$ 0.05).

(vii)   No significant relation of sexual dysfunction was observed with duration (P $>$ 0.5), severity (P $>$ 0.95) or mode of treatment (P $>$ 0.05) of diabetes.

(viii)   Duration of diabetes had no effect on its severity (P $>$ 0.975).

(ix)   Neuropathy was the more frequent (79.7%) complication of diabetes than the vasculopathy (31.3%) and most of the patients with vasculopathy (90.0%) had neuropathy too. But both these complications were independent of each other (P $>$ 0.1).

(x)   In the presence of neuropathy (either peripheral or autnomomic) the frequency of sexual disturbances was found to be high (78.4%) and both were closely associated ($P < 0.01$).

(xi)   The occurrence of neuropathy had no relation with the age of patients ($P > 0.1$) or severity of hyperglycaemia ($P > 0.25$) while there was a definite relation with duration of diabetes, the frequency getting higher with increasing duration ($P < 0.05$).

(xii)   Sexual disturbances in diabetics were found to be irreversible as no patient except one improved with the control of diabetes. This case too had very minimal subjective improvement.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Abelson, D. (1975) Diagnostic value of the penile pulse and blood pressure : a Doppler study of impotence in diabetics. J. Urol., 113, 636-39.

2. Adelson, E.R. (1974) Brief guide to office counselling : Premature ejaculation. Med. Aspects Human Sex, 8, 83-84.

3. Ahuja, M.M.S., Sivaji, L., Garg, V.K. and Mitroo, P. (1972) Prevalence of diabetes in Northern Indian (Delhi area). Horm. Metab. Res., 4, 321.

4. Alam, S.M., Chandra, R., Mahendru, R.K., Chandra, S. and Govil, M.K. (1981) Sexual dysfunction in male diabetics. Indian Medical Gazette, 115, 12-13.

5. Andaloro, V.A. and Dube, A. (1975) Treatment of retrograde ejaculation with brompheniramine. Urology, 5, 520-522.

6. Bartak, V., Josifko, M. and Horackova, M. (1975) Juvenile diabetes and human sperm quality. Int. J. Fertil., 20, 30.

7. Bell, E.T. (1952) A postmortem study of vascular disease in diabetics. Arch. Path (Lab.Med.), 53, 444-55.

8. Berquist, N. (1954) The gonadal function in male diabetes. Acta Endocr., 18 (Suppl), 3-29.

9. Berry, J.N., Malik, K. and Gupta, H.D. (1966) prevalence of postprandial glycosuria in an urban community. J. Ass. Phys. Ind., 14, 519.

10. Bhatia, S.G., Sainani, G.S., Nayak, N.J. and
    Diwate, P.G. (1976) Valsalva manoeuver as
    a test of autonomic neuropathy in diabetes
    mellitus. J. Ass. Phys. Ind., 24, 89-93.

11. Bhu, N. and Bharadwaj, T.P. (1969) Diabetic
    neuropathy. J. Ass. Phys. Ind., 17, 587.

12. Bobs, E. and Comarr, A.E. (1960) Neurological
    disturbances of sexual function with special
    reference to 529 patients with spinal cord
    injury. Urol. Survey, 10, 191-222.

13. Bomford, R.R. and Mason, A.S. (1973) Diabetes
    mellitus. In Price's Textbook of the Practice
    of Medicine, ed. Scott, R.B., 11th edition,
    496-512. Oxford University Press, London.

14. Bourne, R.B., Kretzschmar, W.A. and Esser, J.H.
    (1971) Successful artificial insemination
    in a diabetic with retrograde ejaculation.
    Fertil. Steril., 22, 275-277.

15. Butterfield, W.J.H. (1964) Bedford diabetes
    survey. Proc. Royal Soc. Med., 57, 196.

16. Campbell, I.W. and McCulloch, D.K. (1979) Marital
    problems in diabetics. The Practitioner,
    223, 343-47.

17. Clawson, B.J. and Bell, E.T. (1949) Incidence of
    fatal coronary disease in non-diabetic and
    in diabetic persons. Arch. Path., 48, 105-6.

18. Collens, W.S., Rabiner, A.M., Zilinskey, J.D. and
    Boas, I.C. (1950) Am. J. Med. Sc., 219, 482.

19. Cooper, A.J. (1972) Diagnosis and management of
    "endocrine impotence". Brit. Med. J., 2,
    34-36.

20. Cudworth, A.G. (1976) The aetiology of diabetes.
     Brit. J. Hosp. Med., 16, 207-216.

21. Cudworth, A.G. (1978) Type I Diabetes Mellitus.
     Diabetologia, 14, 281-291.

22. de Wardner, H.E. (1969) The Kidney, 3rd ed. ELBS,
     London, 320.

23. De Zoysa, V.P. (1951) Clinical variations of the
     diabetic syndrome in a tropical country,
     Ceylon. Arch. Intern. Med., 88, 812.

24. Dolman, C.L. (1963) Diabetes and its complications.
     Neurology, 13, 135.

25. Dyrberg, T., Benn, J., Christiansen, J.S., Hilsted,
     J. and Nerup, J. (1981) Prevalence of diabetic
     autonomic neuropathy measured by simple bedside
     tests. Diabetologia, 20, 190-4.

26. Editorial (1974) Diabetic autonomic neuropathy.
     Brit. Med. J., 2, 2-3.

27. Eliasson, R., Wide, L. and Wiklund, B. (1970)
     Sexual function in male diabetic patients.
     Excerpta Medica I.C.S., 5, 147.

28. Elisberg, E.I. (1963) Heart rate response to the
     valsalva maneuvre as a test of circulatory
     integrity. J.A.M.A., 186, 202-205.

29. Ellenberg, M. and Weber, H. (1966) Retrograde
     ejaculation in diabetic neuropathy. Ann.
     Intern. Med., 65, 1237-46.

30. Ellenberg, M. (1970) Significance of diabetic
     neuropathy in impotence. Diabetes, 19, 384.

31. Ellenberg, M. (1971) Impotence in diabetes : the
     neurological factor. Ann. Intern. Med., 75,
     213-19.

32. Ellenberg, M. (1980) Development of urinary
    bladder dysfunction in diabetes mellitus.
    Ann. Intern. Med., 92, 321-3.

33. Ellenberg, M. and Weber, H. (1967) The incipient
    asymptomatic diabetic bladder. Diabetes, 16,
    331-35.

34. Ewing, D.J., Campbell, I.W., Burt, A.A. and
    Clarke, B.F. (1973) Vascular reflexes in
    diabetic autonomic neuropathy. Lancet, 2,
    1354-56.

35. Ewing, D.J., Campbell, I.W. and Clarke, B.F. (1981)
    Heart rate changes in diabetes mellitus.
    Lancet, 1, 183-6.

36. Ewing, D.J., Campbell, I.W., Murray, A., Neilson,
    J.M.M. and Clarke, B.I. (1978) Immediate
    heart - rate response to standing : simple
    test for autonomic neuropathy in diabetes.
    Brit. Med. J., 1, 145-147.

37. Faerman, I., Glocer, L., Fox, D., Jodzinsky, M.N.
    and Rapaport, M. (1974) Impotence and diabetes:
    histological studies of the autonomic nerve
    fibres of the corpora cavernosa in impotent
    diabetic males. Diabetes, 23, 971-976.

38. Faerman, I., Jadzinsky, M. and Podolsky, S. (1980)
    Diabetic neuropathy and sexual dysfunction.
    In Clinical Diabetes : Modren Management, ed.
    Podolsky, S., New York : Appleton - Century -
    Crofts, 293-340.

40. Fairburn, C.G. (1981) The sexual problems of diabetic
    men. Brit. J. Hosp. Med., 25, 484-91.

41. Fairburn, C.G., Wu, F.C.W., McCulloch, D.K.,
       Borsey, D.Q., Ewing, D.J., Clarke, B.F.
       and Bancroft, J.H.J. (1982) The clinical
       features of diabetic impotence : A
       preliminary study. Brit. J. Psychiat.,
       140, 447-452.

42. Fisher, C., Gross, J. and Zuch, J. (1965)
       Diabetic impotence. Arch. Gen. Psychiatry,
       12, 29.

43. Foster, D.W. (1980) Diabetes mellitus. In Harrison's
       Principles of Internal Medicine. ed. Isselbacher,
       K.J., Adams, R.D., Braunwald, E., Petersodorf,
       R.G. and Wilson, J.D., Tokyo : McGraw - Hill
       Kogakusha Ltd., 1741-1755.

44. Fox, C.A., Ismail, A.A.A., Love, D.N., Kirkham,
       K.E. and Loraine, J.A. (1972) Studies on
       the relationship between plasma testosterone
       levels and human sexual activity. Journal
       of Endocrinology, 52, 51-58.

45. Frank, E., Anderson, C. and Rubinstein, D. (1978)
       Frequency of sexual dysfunction in 'normal'
       couples. New Eng. J. Med., 299, 111-15.

46. Friedman, M. (1973) Success phobia and retarded
       ejaculation. Am. J. Psychother., 27, 78-84.

47. Gahlaut, D.S. and Sharma, S.N. (1982) Erectile
       impotence in male diabetics. J. Ass. Phys.
       Ind., 30, 71-72.

48. Gaskell, P. (1971) The importance of penile blood
       pressure in cases of impotence. Canad. Med.
       Assoc. J., 105, 1047-51.

49. Gebhard, P.H.and Johnson, A.B. (1979) Marginal
    tabulations of the 1938-1963. Interviews
    conducted by the Institute for Sex Research.
    Philadelphia : W.B. Saunders.

50. Goldenberg, S., Alex, M. and Blumenthal, H.T.
    (1958) Sequelae of arteriosclerosis of the
    aorta and coronary arteries. Diabetes, 7,
    98-108.

51. Gupta, O.P., Dave, D.M., Rawal, M.L., Sutaria,
    V.M., Bodiwala, N.K., Parikh, N.K., Shah,
    B.S., Gupta, P.S., Joshi, M.N. and Agarwal,
    S.B. (1970) A study of the prevalence of
    diabetes mellitus by means of a house to
    house survey in Ahmedabad (Gujarat).
    Excerpt. Med. Intern. Cong. Series, 221, 6.

52. Hastings, D.W. (1975) Sexual potency disorders
    of the male. In Comprehensive Text book of
    Psychiatry (ed. A.M. Freedman and H.I. Kaplan).
    1072-76.
    Baltimore : Williams and Wilkins.

53. Herman, A., Adar, R. and Rubinstein, Z. (1978)
    Vascular lesions associated with impotence
    in diabetic and nondiabetic arterial
    occlusive disease. Diabetes, 27, 975-81.

54. Horstmann, P. (1950) The excretion of androgens
    in human diabetes mellitus. Acta Endocr.
    (Kobenhayn), 5, 261.

55. Hosking, D.J., Bennett Hampton, J.R., Evans, D.F.,
    Clark, A.J. and Robertson, G. (1979) Diabetic
    impotence : studies of nocturnal erection
    during REM sleep. Brit. Med. J., 2, 1394-96.

56. Jadzinsky, M., Faerman, I. and Fox, D. (1973) Neuropatia diabetica visceral. Acta Diabetologica Latina, 10, 208-260.

57. Johnson, J. (1968) Disorders of Sexual potency in the Male. Oxford : Pergamon Press.

58. Joslin, E.P. (1959) The Treatment of Diabetes Mellitus, ed. Joslin, E.P., Root, H.F., White, P. and Marble. A., 211, Phildelphia : Lea and Febiger.

59. Kaplan, H.S. (1974) The New Sex Therapy. New York: Brunner/Mazel, 289-315.

60. Kaplan, H.S., Kohl, R.N. and Pomeroy, W.B. (1974) Group treatment of premature ejaculation. Arch. Sex. Behav., 3, 443-452.

61. Karacan, I., Salis, P.J., Ware, J.C., Scott, F.B. and Beutler, L.E. (1966) Diabetic impotence. Arch. Gen. Psychiatry, 15, 183.

62. Keen, H. (1959) Autonomic neuropathy in diabetes mellitus. Postgrad. Med. Jour., 35, 272-80.

63. Kent, J.R. (1966) Gonadal function in impotent diabetic males. Diabetes, 15, 537-38.

64. Khandelwal, P.D., Kothari, L.K., Bhu, N. and Bordia, A. (1981) Effect of diabetes mellitus on male fertility. J. Ass. Phys. Ind, 29, 139-42.

65. Kim, H.S., Meister, L. and Brunjes, S. (1966) The autonomic nervous system in impotent diabetic males. Diabetes, 15, 537-38.

66. Kinsey, A.C., Pomeroy, W.B. and Martin, C.E. (1948) Sexual Behaviour in Human Male. Philadelphia, W.B. Sauders Company, 193-217.

67. Klebanow, D. and MacLeod, J. (1960) Semen quality
    and certain disturbances of reproduction in
    diabetic men. Fertil. Steril., 11, 255-61.

68. Koch, K. (1910) Testes in diabetes mellitus.
    Virchows Arch. Path. Anat. 202, 376.

69. Kolodny, R.C., Kahn, C.B., Goldstein, H. and
    Barnett, D.M. (1974) Sexual dysfunction
    in diabetic men. Diabetes, 23, 306-9.

70. Kolodny, R.C., Masters, W.H. and Johnson, V.E.
    (1979) Male sexual dysfunction in diabetes.
    In Text book of Sexual Medicine, 1st ed.,
    128-136, Little, Brown and Co., Boston.

71. Krosnick, A. and Podolsky, S. (1981) Diabetes and
    sexual dysfunction : restoring normal ability.
    Geriatrics, 36, 92-100.

72. Laurence, D.G. and Locke, S. (1963) Diabetic
    complications. Brit. Med. J., 1, 784.

73. Learmouth, J.R. (1931) A contribution to the
    neurophysiology of the urinary bladder in
    man. Brain, 54, 147-176.

74. Lester, E., Grant, A.J. and Woodroffe, F.J.(1980)
    Impotence in diabetic and non-diabetic
    hospital outpatients. Brit. Med. J., 281,
    354-55.

75. Levine, S.B. (1976) Marital sexual dysfunction :
    ejaculation disturbances. Ann. Intern. Med.,
    84, 575-79.

76. Locke, S. (1953) Diabetes. Med. Clin. N. Amer.
    49, 1081.

77. Machleder, H.I. (1978) Sexual dysfunction in
     aortoiliac occlusive disease. Human Sexuality,
     May, 125-126.

78. Magee, M.C. and Fried, F.A. (1978) Neurology for
     the urologist. J. Cont. Ed. Urol., 17, 11-36.

79. Malins, J.M. (1966) Diabetes mellitus. ed. Duncan,
     L.J.P. University of Edinburgh Pfizer Medical
     Monographs, 137-143, Edinburgh University Press.

80. Malins, J.M. (1968) The eye in diabetes. In Clinical
     diabetes Mellitus. 186-210, London : Eyre and
     spottiswoode.

81. Marmor, J. (1975) Impotence and ejaculatory
     disturbances. In Comprehensive Text book
     of Psychiatry. (ed. A.M. Freedman and H.I.
     Kaplan) 2nd ed., 1525. Baltimore : Williams
     and Wilkins.

82. Martin, M.M. (1953) Involvement of autonomic nerve
     fibres in diabetic neuropathy. Lancet, 1,
     560-65.

83. Masters, W.H. and Johnson, V.E. (1970) Human
     sexual Inadequacy. Boston : Little, Brown
     & Company, 92-115.

84. May, A.G., DeWeese, J.A. and Rob, C.G. (1969)
     Changes in sexual function following
     operation on the abdominal aorta. Surgery,
     65, 41-47.

85. Mayne, N. (1965) Diabetic neuropathyl Lancet,
     2, 1313.

86. McFadgean, A.J.S. and Yeung, R. (1968) Diabetic
     neuropathy. Diabetes, 17, 224.

87. Michal, V., Kramar, R. and Pospichal, J.(1974)
    Femoropudendal bypass, internal iliac
    thromboendart erectomy and direct arterial
    anastomosis to the cavernous body in the
    treatment of erectile impotence. J.
    Cardiovasc. Surg. 15, 356-59.

88. Mills, C.A. (1930) Diabetes mellitus, climate-
    responsible factor in the etiology. Arch.
    Intern. Med., 46, 589.

89. Mohan, V. Ramachandran, A., Sivakumar, V.K. and
    Viswanathan, M. (1980) Diabetic autonomic
    neuropathy - clinical evaluation. J. Diab.
    Asso. Ind., 20, 69 - 71.

90. Montague, D.K., James, R.E., de Wolfe, V.G. and
    Martin, L.M. (1979) Diagnostic evaluation,
    classification and treatment of men with
    sexual dysfunction. Urology, 14, 545 - 8.

91. Montenero, P. and Donatone, E. (1962) Diabete
    et activite sexuelle Ehez Thomme. De
    Diabete,8, 327-332.

92. Moses, S.G.P., Seshiah, V. and Prithika (1980)
    sexual dysfunction in diabetic men.
    Preceedings of the fourth National
    Congress on Diabetes, January, 136 - 43.

93. Nakra, B.R.S., Wig, N.N. Varma, V.K. (1977)
    A study of male potency disorders. Ind.
    J. Psychiat., 19, 13 - 18.

94. National Diabetes Data Group (1979) Classification
    and diagnosis of diabetes mellitus and other
    categories of glucose intolerance. Diabetes,
    28, 1039 - 57.

95. Naunyn, B. (1906) Der Diabetes mellitus, ed.
    2, Vienna, Alfred Holder.

96. Nayyar, S. (1966) Inaugral address in World
    Congress on Diabetes in the Tropics,
    Bombay. In Diabetes in the Tropics, ed.
    Patâl, J.C. and Talwalkar, N.G., p. v-vii.

97. Neudorfer, G. (1950) Generativer Ablauf bei
    Zuckerkrarken mannlichen. Partnem. Inaugural
    Dissertation, Munich.

98. Nies, A.S. (1972) Clinical Pharmacology. Ist edition,
    Eds. Melmon, K. and Morelli, H., Macmillan and
    Co., New York, 143 - 147.

99. Noronha, J.L., Bhandarker, S.D., Shenoy, P.N. and
    Retnam, V.J. (1981) Autonomic neuropathy in
    diabetes mellitus. J. Postgrad. Med., 27, 1-6.

100. Oakley, W.G. (1949) In discussion of Strauss,
     E.B., reference 119.

101. Ostrander, L.D., Francis, T., Hayner, N.S.,
     Kjelsberg, M.O. and Epstein, F.H. (1965)
     The relationship of cardiovascular disease
     to hyperglycaemia. Ann. Intern. Med., 62, 1188.

102. Patel, J.C. and Dhirwani, M.K. (1966) Incidence
     of diabetes in Bombay. J. Ind. Med. Sci.,
     12, 10.

103. Paterson, J.W.,Kohner, E.M., Dolley, C.T. and
     Oakley, N.W. (1967) Arterial fluorescein
     studies in diabetic retinopathy. Diabetes,
     16, 1.

104. Pódolsky, S. (1971) Lipoatrophic diabetes and
     miscellaneous conditions related to diabetes
     mallitus. In Joslin's Diabetes Mellitus
     (Ed. 11) Marbel, A., White, P. and Bradley,
     R.F. (editors). Philadelphia, Lea and Febiger.

105. Pyke, D.A. (1968) The incidence and prevalence of
     diabetes. In Clinical Diabetes and its
     Biochemical Basis, ed. Oakley, W.G., Pyke,
     D.A. and Taylor, K.W., 181-209, Oxford and
     Edinburgh : Blackwell Scientific publications.

106. Rackow, F. (1968) Diabetic neuropathy. In Clinical
     Diabetes and its Biochemical Basis. ed. Oakley,
     W.G., Pyke, D.A. and Taylor, K.W., Oxford :
     Blackwell Scientific publications, 542-561.

107. Renshaw, D.C. (1975) Impotence in diabetes.
     Diseases of the Nervous System, 36, 369-71.

108. Renshaw, D.C. (1979) Diabetic impotence -
     inevitable or imposed, Brit. J. Sexual
     Med., 6, 48-51.

109. Rivarola, M.A., Faerman, I. and Jadzinsky,M.
     (1970) Plasma testosterone levels in diabetic
     men with and without normal sexual potency.
     Excerpta Medica. I.C.S., 5, 38.

110. Rubin, A. and Babbott, D. (1958) Impotence and
     diabetes mellitus. J.A.M.A., 168, 498-500.

111. Rundles, R.W. (1945) Diabetic neuropathy. Medicine
     (Balt), 24, 111-160.

112. Salzman, L. (1962) Developments in Psychoanalysis.
     New York, Grune and Stratton, 167 - 176.

113. Satyanarayan, R.P., Naik, B.K., Saboo, R.V.,
      Ramachandran. A., Dandelia, P.B. and Parley,
      K. (1966) Indidence of diabetes in Hyderabad.
      Proc. World Cong. on Diabetes in Tropics.
      Ed. Patel, J.C. and Talwalkar, N.G., Bombay,
      68 - 75.

114. Schapiro, B. (1943) Premature ejaculation :
      Review of 1130 cases. J. Urol., 50, 374.

115. Schatz, I.J., Podolsky, S. and Frame, B. (1963)
      Idiopathic orthostatic hypotension, J.A.M.A.,
      186, 537 - 540.

116. Schiavi, R.C. and Hogan, B. (1979) Sexual problems
      in diabetes mellitus : Psychological aspects,
      Diabetes care, 2, 9-17.

117. Schoffling, K. (1970) Diabetes mellitus and
      male gonadal function. Presented at VII
      Congress of International Federation of
      Diabetes, Buenos Aires, Argentina, August,
      1970.

118. Schoffling, K., Federlin, K., Digschuneit, H.
      and Pfeiffer, E.F. (1963) Disorders of
      sexual function in male diabetics. Diabetes,
      12, 519-27.

119. Scott, F.B., Fishman, I.J. and Light, J.K. (1980)
      An inflatable penile prosthesis for treatment
      of diabetic impotence. Ann. Intern. Med.,
      92, 340-2.

120. Shukla, G.D., Srivastava, O.N., and Katiyar, B.C.
      (1979) Sexual disturbances in temporal lobe
      epilepsy : a controlled study. Brit. J.
      Psychiat., 134, 288 - 92.

121. Simpson, S.L. (1950) Impotence, Br. Med. J.,
     1, 692 - 97.

122. Strauss, E.B. (1949) Impotence. Med. Soc.
     Trans., 64, 291-299.

123. Strauss, E.B. (1950) Impotence from the
     Psychiatric standpoint. Brit. Med. J.,
     1, 697-99.

124. Tattersall, R. (1982) Sexual problems of
     diabetic men. Brit. Med. J., 285, 911-12.

125. Taylor, R.W. (1975) Some of the commoner sexual
     disorders : problems affecting mainly the
     male. Brit. Med. J., 2, 739-741.

126. Tripathy, B.B. (1971) Population survey for
     detection of frank and latent diabetes
     in one part of Cuttack, Orissa. J. Ind.
     Med. Asso., 54, 55.

127. Viswanath, I., Bajpai, H.S. and Katiyar, B.C.
     (1976) Neurological complications in
     diabetes mellitus. J. Ind. Med. Asso.,
     66, 176-80.

128. Vijayan, G., Singh, N., Roy, S. and Pathak,S.N.
     (1971) Diabetic complication. Ind. J. Med.
     Res., 59, 1846.

129. Von Noorden, C. (1903) Die Zuckerkrankheit und
     thre Behandlung. Berlin, August Hirschwald.

130. Wagner, R., White, P. and Bogan, I.K. (1942)
     Sexual development in juvenile diabetics.
     Am. J. Dis. Child, 63, 667.

131. Warren, S. and Le Compte, P.M. (1952) The
       pathology of Diabetes Mellitus (ed. 3).
       Philadelphia, Lea and Febiger.

132. Weiss, H.D. (1972) The physiology of human penile
       erection. Ann. Intern. Med., 76, 793-99.

133. Whalen, G.E., Soergel, K.H. and Geenen, J.E.
       (1969) Diabetic dirrhoea. Gastroenterology,
       56, 1021 - 1027.

134. Wilkerson, H.L.C. and Krall, L.P. Jr. (1947)
       Diabetes in New England Town. J.M.M.A.,
       135, 209.

135. Wilson, J.L. and Marks, J.H. (1951) Calcification
       of Vas Deferens : Its relation to diabetes
       mellitus and arteriosclerosis. New Eng. J.
       Med., 245, 321-325.

136. Wingrade, A.I. (1972) Diabetic neuropathy.
       N. Eng. J. Med., 286, 1261.

APPENDICES

APPENDIX - I

CRITERIA FOR DIAGNOSIS OF DIABETES MELLITUS

(National Data Group of American Diabetes Association, 1979)

Any one of the following are considered diagnostic of diabetes :

A. Presence of the classic symptoms of diabetes, such as polyuria, polydipsia, ketonuria and rapid weight loss, together with gross and unequivocal elevation of plasma glucose.

B. Elevated fasting glucose concentration on more than one occasion :

Venous whole blood

$>$ 120 mg/dl (6.7 mmol/L)

If the fasting glucose concentration meets these criteria, the OGTT is not required.

C. Fasting glucose concentration less than that which is diagnostic of diabetes (B, above), but sustained elevated glucose concentration during the OGTT on more than one occasion. Both the 2-h sample and some other sample taken between administration of the 75-g glucose dose and 2-h later must meet the following criteria :

Venous whole blood

$>$ 180 mg/dl (10.0 mmol/L).

PROFORMA

Case No.

O.P.D./Wd. and Bed. :

Date of interrogation :

Date of admission :

Date of discharge :

Name :   Age :

Address :   Height: Weight :

Occupation :   Education :

Marital status: Single/Married/Widowed/Divorced.

PRESENTING SYMPTOMATOLOGY WITH DURATION

1.

2.

3.

4.

5.

FAMILY HISTORY

Joint Family/Unitary Family

No. of family members :

Duration of married life :   Age of wife:

No. of children :

        Before onset of diabetes -

        After onset of diabetes -

Age of last child :

PERSONAL DATA AND PAST HISTORY

Intake of alcohol :   Smoking :

Intake of drugs (Antihypertensives, antidepressants,

　　　　　　　Phenothiazines, Oestrogens)

History of Mumps/Trauma/Operation/intermittent

claudication.

DIABETES

(1) Type :   Type I/Type II

　　　　　Symptomatic/Asymptomatic

(2) Duration :   Age at onset of diabetes:

(3) How diabetes was detected :

(4) Past Treatment :- Diet/Insulin/Oral drugs/

　　　　　　　Insulin + Oral drugs

　　　　　　　Specific drugs and dose

　　　　　　　Continued / Interrupted

(5) Controlled/Uncontrolled

SEXUAL STATUS

(1) Any decline from earlier level of performance: Yes/No

　　(a) Onset : acute/insidious

　　(b) Progress : Progressive/stationary/regressive

　　　　　　　- With treatment

　　　　　　　- Without treatment

(2) Sexual dysfunction

    (a) antedated/coincided/followed the onset
        of diabetes.

    (b) Interval

<u>Subjective assessment of the patient</u>

approx. % of earlier normal level (with duration)	Present level

(3) Libido

    (a) fantasy of sexual nature

    (b) dreams of sexual nature

    (c) nocturnal emissions

    (d) morning erections

    (e) sexual excitement on seeing wife/
                       other famales

    (f) desire for coitus

(4) Degree of erection

    (a) fantasy

    (b) masturbation

    (c) intercourse - with wife
                    - with other female
                    - with male

(5) Ejaculatory time :

<u>Disturbances</u> :

    (a) Failure of ejaculation

    (b) Premature ejaculation

    (c) Retrograde ejaculation

(6) Frequency of sexual outlets

   (sexual intercourse/masturbation)

   If no intercourse, reason for it - no desire

                                    - no erection

                                    - no partner

                                    - any other

(7) Deviation

(8) Patient's attitude towards the declined/absent
    Sexual function

          - Worried

          - Concerned but not worried

          - indifferent

          - happy

(9) Interrogation of sexual partner

(10) Overall sexual functional status

     (a) Normosexual (more than one outlet per month,
                      no decline from earlier level
                      of performance).

     (b) Hyposexual (Less than one outlet per month,
                      definite decline from earlier
                      level, impotence disturbance
                      of ejaculation).

     (c) Hypersexual (excessive indulgence in sexual
                      activity leading to social or
                      domestic problems, embrassment
                      to others etc.).

     (d) Deviations.

(11) Does he indulge in sex

      - Because he feels a strong desire for sex

      - to fulfil a routine

      - on request of his wife

PSYCHIATRIC STATUS

COMPLICATIONS OF DIABETES

NEUROPATHY

A. Peripheral

- Pain	- Absent jerks (ankle)
- Paraesthesia	- Sensory impariment

B. Autonomic

- Pulse : - Resting - Standing	effect of valsalva maneuver
- B.P.	- Postural giddiness
⌈ lying	- nocturnal diarrhoea
⊢ standing	- bladder function
⌊ after Angised	
- Pupillary changes	- residual urine
- Sweating/anhidrosis	

VASCULOPATHY

    - Arterial wall

    - Peripheral pulsations

    - H/O cerebral ischaemia
        intermittent claudication
        myocardial ischaemia
        trophic ulcers/gang-rene

RETINOPATHY

NEPHROPATHY

- Anasarca

- Albuminuria

- Blood Urea/S. creatinine

OTHER SYSTEMS

INVESTIGATIONS

(1) TLC =   DLC =   Hb =   ESR =

(2) Urine :   Sugar   Albumin

(3) B.Sugar :   Fasting   2 hour after glucose/load/meal

(4) B. Urea

(5) S. Cholesterol

(6) Seminogram

(7) E.C.G.

(8) Screening/X-ray chest

(9) V.D.R.L.

(10) Any other

TREATMENT GIVEN

Follow - up

(a) Control of diabetes

(b) Sexual functioning - normal throughout

- improved

- subjective % of improvement

- Continued to ramain impaired

- Worsened

(c) Relation of control of diabetes with sexual functions

- direct

- inverse

- No relation

Summary of the case :

APPENDIX - III

STANDARD HEIGHT AND WEIGHT FOR INDIAN MEN

(Life Insurance Corporation of India)

Height (Feet-inches)	Standard weight (Kg)
5' 0"	-
5' 1"	-
5' 2"	56.3 - 60.3
5' 3"	57.6 - 61.7
5' 4"	58.9 - 63.5
5'5"	60.8 - 65.3
5' 6"	62.2 - 66.7
5' 7"	64.0 - 68.5
5' 8"	65.8 - 70.8
5' 9"	67.6 - 72.6
5' 10"	69.4 - 74.4
5' 11"	71.2 - 76.2
6' 0"	73.0 - 78.5
6' 1"	75.3 - 80.7
6' 2"	77.6 - 83.5
6' 3"	79.8 - 85.7